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16. ABSTRACT

An extensive review of 338 references on the effects of pollution on freshwater fish covering 1976-1977 is presented. These references cover: Reviews and Symposia, Dissolved Gases, pH, Insecticides, Herbicides, Piscicides and Anesthetics, Inorganics, Organics, Chlorine and Wastewater, Pulp and Paper, Radioactive and Physical Factors.

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REVIEWS AND SYMPOSIA

An extensive review of 464 references on the effects of pollution on freshwater fish was presented by Brungs *et al.*[1] Metals, pesticides, and domestic and industrial wastes were included, as well as the effects of water quality (salinity, pH, and O_2) and radioactivity. Papers on recent advances in fish toxicology were compiled in proceedings of an Environmental Protection Agency (EPA) symposium on marine, estuarine, and freshwater quality.[2] The freshwater segment of the symposium addressed the transport and biological modeling capability of EPA laboratories, cold climate aquatic biology, lake trophic states in the eastern U. S., and the impact of toxic substances on freshwater systems. A book entitled "The Ecological Physiology of Fish. Part I" was edited by Stroganov[3] and included summaries of papers submitted to the Third Conference in Kiev (U. S. S. R.), November 1976. Among the problems discussed were physiological and biochemical responses of fish to environmental changes including natural abiotic factors, effects of various toxicants, energy, water and salt metabolism, composition and functions of fat and proteins in different fish tissues. Wells[4] provided a selected bibliography on the effects of several aquatic toxicants to rainbow trout (*Salmo gairdneri*). This bibliography contained over 470 references which were presented in three sections; papers on holding and disease, physiology and effects of toxicants, and author and keyword indexes. An inventory of data on contaminants in aquatic organisms was established as part of a co-operative project of the United Nations Environmental Programme and the Food and Agriculture Organization.[5] The directory contained information from questionnaires sent to marine and freshwater institutions around the world and included indexes to projects by contaminant, host subject, geographical area, country, and detailed project activities.

Several studies concerning the effects of heavy metals in the environment were collected from over 270 papers presented at the International Conference on Heavy Metals in the Environment held in Toronto, Canada in October, 1975.[6] Symposium proceedings were published in three volumes. Volume I contained analytical papers, together with plenary addresses, futures and regulatory papers. Volumes II and III contained pathways and health orientated papers, respectively. The Office of Water Research and Technology, U. S. Department of the Interior,[7–11] compiled a five-volume set of abstracts from the Selected Water Resources Abstracts through February 1977 for Cd, Cr, Co, Cu, Fe, Pb, Mn, Ni, and Zn. Mercury was covered in a separate series.[12] The European Inland Fisheries Advisory Commission Working Party on Water Quality Criteria of European Freshwater Fish,[13] after a critical examination of the available data, proposed tentative criteria for dissolved cadmium at four levels of water hardness for rainbow trout and perch (*Perca fluviatilis*). Hertzberg[14] reviewed recent publications on Hg in fish with respect to Israel and concluded that there were no health hazards involved. The National Research Council Committee[15] on medical and biological effects of environmental pollutants reviewed the current knowledge on the distribution of Cu in the biosphere. A brief summary was pre-

sented of the literature pertaining to the acute and chronic toxicity of Cu to freshwater fish and other aquatic organisms. Selected methods for the quantitative analysis of Cu in water and air, and in biological materials, are described in detail in the Appendix. Black *et al.*[16] prepared an annotated summary of 101 selected papers relating Cu concentrations in water to deleterious effects on aquatic organisms. Tables were arranged by increasing concentration, from 0.01 to 63,500 μg/l, for various species and group of organisms.

A compilation of abstracts of selected projects supported by the Environmental Protection Agency on environmental effects of energy was prepared for the EPA Office of Energy and Minerals by Johnson.[17] The report included the ecological effects of energy and environmental transport processes. Ralston *et al.*[18] compiled a selected bibliography of references with abstracts on the ecological effects of coal strip-mining with particular emphasis on the Northern Great Plains. References were in two parts. Part I contains references and abstracts and Part II a "Keyword in title" index to the references. Pages 131 to 140 contain references to animals and fish, Dewitt *et al.*,[19] Melvin *et al.*[20–22] Melvin and Ehrenspeck,[23] and Melvin and Ross[24–26] provided a series of quarterly reports concerned with all aspects of aquatic and terrestrial oil pollution. The authors reviewed scientific and technical publications, presented citations of published literature and patents, and reported the status of research projects and scheduled oil-related conferences during each quarter.

The absorption and movement, degradation, persistence and toxicity of diquat and endothall in the environment was reviewed by Simsiman *et al.*[27] Both compounds were retained for only a short time in the body after being absorbed by fish. The acute toxicity levels of diquat and endothall to mammals, fish, and invertebrates were well above the recommended dosages for weed control. In addition to a review of the literature on the effects of phenol on fish, Matej[28] studied the effects of phenol on the conditioned reflex activity and the hypothalamic neurosecretion in the guppy (*Poecilia reticulata*). He concluded that physiological responses to phenol are of a defensive-adaptive nature, with the central nervous system showing the highest degree of sensitivity to the toxic action of phenol. Huffman and Desai-Greenaway[29] conducted a comprehensive survey of the world literature regarding the toxicological effects of vinylidene chloride to fish and other aquatic organisms and its fate in the aquatic ecosystem. The authors noted there was little information regarding this subject in the literature. Fickeisen and Schneider[30] edited the proceedings of a workshop wherein results from the many laboratories dealing with gas bubble disease were presented. Included were formal papers as well as summaries of five informal round-table discussions.

A treatise on the many effects of construction activities in wetlands of the U. S. was presented by Darnell.[31] Included in the effects were pertaining to freshwater fishes. Human activities were implicated in the loss of 87 native species of fishes up to 1966. Environmental effects were discussed and literature citations included effects due to flow extremes and consumptive losses. Also covered were effects due to dam construction with losses of stream fauna and mechanical losses with fish passage through and over dams and through turbines. Blahm[32] presented an overview of the adverse effects of water diversions on fish populations of the west and, in particular, the Pacific northwest. Diversions discussed included diversions for irrigation, channelization, and passage through industries with subsequent changes in water quality. Losses were discussed in general terms in most instances but the author suggested that juvenile salmon and trout losses on the Columbia River in any year were as high as 70 to 90%, most of which could be indirectly attributed to diversion within the river system. Collins[33] also presented a general review of the effects of dams in the Columbia River system on the anadromous fishes. Reported effects were blocked access to former areas of habitation, conversion of spawning grounds into sediment-loaded impoundment bottoms, elimination of spring freshets, creation of controlled runoff that delayed smolt arrival to the estuary, elevation of temperature, imposition of mechanical hazards such as turbine passage, and other problems such as supersaturation and untenable zones of passage through stratifications where the surface waters were lethally warm and the hypolimnion too low in DO to support salmon. Some remedial activities were presented with prospects for ameliorating the adverse effects.

WATER QUALITY

Dissolved gases. Isaacson[34] suggested that gas supersaturation was a necessary condi-

tion for gas bubble disease, but the disease itself was triggered by cold stress. Tiger barbs subjected to cold stress showed a high incidence of pop eye, a symptom of gas bubble disease. In studies of gas bubble disease, Nebeker and Brett[35] calculated threshold values of 114.5, 113.5, and 114% saturation for coho salmon (*Oncorhynchus kisutch*), sockeye salmon (*Oncorhynchus nerka*), and steelhead (*Salmo gairdneri*), respectively. Calculations resulted in 96 hour LC50 values of 120.5, 116.7, and 116.0% total gas saturation for the same fish. In addition, Nebeker *et al.*[36] determined that 114% gas saturated water was near the lethal threshold for adult sockeye salmon in 60 cm of water at 12°C. It was indicated that adult sockeye salmon migrating through the Columbia River would not be affected by gas bubble disease if total gas saturation levels in the river remained below 115%. Weber and Schiewe[37] compared normal lateral line response of juvenile steelhead trout to a standardized set of stimuli with the response of fish affected by gas bubble disease. The ability of the fish to respond to stimuli was either diminished or lost entirely as gas emboli formed in the scale pockets of the lateral line. This sensory loss was demonstrated to be reversible. Otto[38] determined gas supersaturation tolerance levels for yellow perch (*Perca flavescens*) and rainbow trout in laboratory studies. Saturation levels as high as 115 and 110% did not affect yellow perch or rainbow trout, respectively. The 8-day median lethal saturation level for yellow perch was 126%, and for rainbow trout 119%.

Thillart *et al.*[39] determined that goldfish were tolerant to anoxia and could survive 10-hour periods of complete anoxia at 20°C without any problem. Increases in the concentration of alanine, creatine, total CO_2, and ADP were demonstrated. Concentrations of ATP and creatine-phosphate decreased with anoxia while AMP, glutamate and volatile fatty acids did not change significantly. Hamor and Garside[40] found that survival of Atlantic salmon (*Salmo salar*) embryos during embryogenesis and during the hatching period was limited primarily by O_2 supply and secondarily by water exchange. Temperature effect was third in rank with lower temperatures reducing the rate of development and enhancing survival through hatching. A study by Guliduv and Popova[41] showed that increasing the O_2 concentration from 9.2 to 17.6 mg/l decreased egg mortality rate and increased the total number of hatched embryos of the bream (*Abramis brama*). Further increase of the O_2 concentration to 33.7 mg/l reversed the trend. A decrease was recorded in the erythrocyte counts in vessels of embryos exposed to supersaturated conditions. Plumb *et al.*[42] discovered that channel catfish (*Ictalurus punctatus*), after 6 days of extremely low O_2 concentration, developed sterile lesions in the skin and muscles. *Aeromonas hydrophila* was isolated from either the lesions or internal organs of moribund fish necropsied the day after the lesions were found. It was hypothesized that hypoxia occurred in peripheral areas of the muscles and internal organs of the fish and these areas became necrotic. The resistance of the fish was lowered to the point that infection of the lesions by the bacterium occurred. Death was attributed to low O_2 concentration and not the bacterium. Health of the surviving fish improved as water quality improved.

Harichaux and Gorin,[43] using a continuous polarographic recording of the respiratory function of *Salmo trutta fario*, showed a variation of O_2-dependence rate, according to previous poisoning in the environment. Continuous measurements of long duration (1 to 2 weeks) showed a cyclic variation of the respiratory activity of *Salmo irideus*. Pegg *et al.*[44] indicated that cyclic fluctuation in the O_2 consumption rate of the brown bullhead (*Ictalurus nebulosus*), the pumpkinseed (*Lepomis gibbosus*), and the bluegill (*Lepomis macrochirus*) was altered in both frequency and amplitude by the presence of acid water. Feldmeth and Baskin[45] determined that the unarmored stickleback showed respiratory independence over a broad range of O_2 concentrations with greatly increased ventilation rates as the O_2 concentration decreased to approximately 1.3 mg/l. Ventilation rate rapidly decreased at lower concentrations. Vigers and Maynard[46] described a rapid bioassay procedure for predicting the toxicity of effluents to rainbow trout. The procedure depends upon the consumption of available O_2 by fish in sealed containers and required less than 8 hours to complete. Threshold concentrations obtained using sodium pentachlorophenate, three kraft mill bleach plant effluents and chloralkali plant effluent, were directly comparable to 96-hour LC50 values obtained using standard methods. Peterka and Kent[47] measured fluctuations of DO and temperature in the natural spawning sites of northern pike (*Esox lucius*), bluegill, and pumpkinseed. Average daily fluctuations were 3.0 mg/l DO and 1.6°C at the

northern pike sites and 4.4 mg DO/l and 3.3°C at the bluegill and pumpkinseed sites. In field experiments with sac larvae of northern pike and embryos and sac larvae of bluegills and smallmouth bass (*Micropterus dolomieui*), tolerance for low DO concentrations decreased from embryo to larval stages. Giles and Vanstone[48] demonstrated that three electrophoretically distinct hemoglobin patterns consisting of 18 different polymorphs occurred during the life cycle of coho salmon. However, the fish did not directly modify their hemoglobin polymorphs in response to variations in water temperature, DO concentration, or salinity. Romanenko and Krisal'nyj[49] showed in chronic experiments with carp (*Cyprinus carpio*) that exposure to elevated concentrations of free CO_2 (0.4, 0.8, and 1.1 mM/l) caused changes in the biocarbonate buffer blood system and in the ion exchange.

Johnson and Sanders[50] determined the acute toxicity of four chemical forest fire retardants, Fire-Trol 100 and 931 (ammonium sulfate or polyphosphate with an attapulgite clay thickener) and Phos-Chek 202A and 259 (diammonium phosphate with a guar gum derivative thickener), to fry and fingerling coho salmon and rainbow trout, fingerling fathead minnows (*Pimephales promelas*), bluegills and largemouth bass (*Micropterus salmoides*), and mature scuds. Both Phos-Chek formulations were more acutely toxic to all life stages of fish and to the scuds than either of the Fire-Trol formulations. Unionized ammonia was believed to be the most toxic component of the fire retardants. Seleši and Vámoš[51] indicated that the lethal concentration of unionized ammonia (NH_3) ranged from 0.2 to 0.5 mg/l. The lethal amount of ammonia was not constant but depended upon the O_2 content of the water. Ammonia acted on the respiratory system and an abundant O_2 supply afforded protection against its paralyzing effect. Steffens[52] determined that free ammonia was strongly toxic to rainbow trout and that optimum production conditions for the rainbow trout only existed when the free ammonia content in the water was <0.01 mg NH_3/l. Low ammonia concentrations not leading to death over long periods increased bacterial disease problems. Concentrations of unionized ammonia ranging from 0.05 to 0.37 mg/l (as NH_3-N) were found by Burkhalter and Kaya[53] to have no effect on mortality or incubation period of rainbow trout eggs. Some retardation in early growth and development occurred at 0.05 mg NH_3-N/l; 0.1 mg/l caused more severe effects through the 42-day test period. The estimated incipient LC50 for rainbow sac fry was 0.25 mg NH_3-N/l.

Guerin-Ancey[54] found that ammonia and urea excretion by *Dicentrarchus labrax* decreased to a constant during a 7-day starvation period. Endogenous Nitrogen Excretion (ENE) was 72 mg N/kg/24 hour of ammonia and 12 mg N/kg/24 hour of urea. ENE was the same for three age groups and occurred later at 16°C than at 20°C. The percentage of urea excreted increased during the starvation period. Nitrogen reserves were calculated to be greater in younger fish and in autumn. Guerin-Ancey[55] also determined that *Dicentrarchus labrax* excreted less ammonia-nitrogen when ambient ammonia was increased and water volume was decreased. Increased ambient ammonia levels corresponded to an increase in the rate of urea excretion. Ammonia poisoning occurred at 10 mg/l. The minimum non-autotoxic volume for bass was 12 l/kg at 16°C, 15 l/kg at 18°C and between 15 and 20 l/kg at 20°C. Ito[56] demonstrated direct absorption of ^{15}N from water by common carp, crucian carp, and loach fry exposed to labeled ammonia. The rate of ^{15}N in the body N increased exponentially with the water concentration of ammonia and the time exposed for the two carp and linearly for the loach. In the case of NO_3-^{15}N, less ^{15}N was absorbed and no apparent correlation was observed between the rate of replacement and the concentration in the water. Within 48 hours after the fry were returned to ammonia-free water, 50% of the absorbed ^{15}N was excreted. The relationships between blood sugar concentration and O_2 saturation of water and between blood sugar concentration and ammonium ion for freshwater fish, *Labeo capensis*, were studied by Hattingh.[57] Blood sugar concentration was increased by increased ammonium ion concentrations and by decreased O_2 saturation of water.

Smith *et al.*[58] determined that the 96-hour LC50 for laboratory-cultured juvenile fathead minnows at 20°C was 0.0243 mg H_2S/l. Fry were twice as sensitive. Response with temperature in wild-stock juveniles varied from a 96-hour LC50 of 0.7754 mg/l at 6.5°C to 0.0423 mg H_2S/l at 25°C. Adverse effects on growth, survival, and fecundity occurred above 0.004 mg H_2S/l, in chronic exposure, to two generations of laboratory-cultured fish stock. Adverse effects on growth and survival occurred above 0.008 mg H_2S/l, in chronic exposures, to wild stock exposed to chronic levels of H_2S up to 346 days. At comparable temperatures apparent safe

levels with long-term exposure were 5- to 7-fold lower than the 96-hour LC50 for both stocks.

Koenst *et al.*[59] exposed brook trout (*Salvelinus fontinalis*) to various concentrations of HCN beginning with adults 144 days prior to spawning and continued through a 90-day growth period of the second generation. The no-adverse-effect level on growth of brook trout after hatch was between 0.0221 and 0.0332 mg HCN/l. Based on survival, the no-adverse-effect level was between 0.044 and 0.055 mg HCN/l. Reproduction was the most sensitive life stage. The maximum acceptable toxicant concentration (MATC) for the brook trout was found to be between 0.0057 and 0.0112 mg HCN/l.

pH. The Norwegian research program entitled Acid Precipitation-Effects on Forest and Fish (the SNSF-project) was briefly discussed by Overrein.[60] The project was initiated as a cooperative effort between Norwegian research institutes to establish the effects of acid precipitation due to transported air pollutants on soil, vegetation, forest, water, and freshwater fish. To illustrate the acidification problem in water courses of southern Norway, it was pointed out that measurements taken during the spring thaws of 1974 and 1975 showed pH levels in the vicinity of 3.0 in run-off water from snowfields, and that fish collected from the field and placed in such water died within a few hours. Schofield[61] summarized experimental investigations and physiological studies, primarily from Norway and Sweden where the problem is particularly severe, concerning the effects on fish of acid precipitation. Documented case studies indicated that extinction of fish populations was often a result of chronic reproductive failure due to acidification-induced effects on sensitive life history stages. Mortality has also been observed due to acid stress (pH 4 to 5) in lakes and streams as a result of interference with the physiological mechanisms regulating active ion exchange across gill membranes, a process that has been demonstrated in the laboratory and field-verified in cases of mass fish mortality in Norway. Beamish[62] reviewed and discussed the effects of acid precipitation on Canadian lakes and fishes, primarily in the mining and smelting region around Sudbury, Ont. Acid precipitation due to airborne sulfur oxides over a period of several decades affected the rate of acidification in many lakes and water chemistry changes included elevated heavy metal concentrations, higher levels of H and SO_4 ions and reduction of alkalinity. Many species of fish were eliminated and reproduction appeared to be the most sensitive major physiological process. The approximate pH range at which fish failed to reproduce in the LaCloche Mountain lakes was >6.0 to 5.5 for smallmouth bass (*Micropterus dolomieui*) and walleye (*Stizostedion vitreum*), and 5.5 to 5.2 for lake trout (*Salvelinus namaycush*). The white sucker (*Catostomus commersoni*) ceased reproduction at pH 5.2 to 4.7. Cyprinids were the most resistant. The lake chub (*Couesius plumbeus*) was observed to reproduce at pH 4.5.

A study conducted by Hoehn and Sizemore[63] chemically characterized three acid mine drainage streams entering Chestnut Creek in southeastern Virginia and assessed biological and chemical alteration in the creek. *In situ* bioassays showed that Chestnut Creek below the affected streams was not lethal to bluegills (*Lepomis macrochirus*) exposed for 192 hours and snails (*Goniobasis* sp.) survived 96 hours before they began to die. However, undiluted acid stream water killed all fish within 11 hours. Absence of fish in Chestnut Creek below the most downstream influence was probably the result of lack of food, unsuitability of the flocculant-covered bottom substrate and, possibly, the irritating nature of water caused by dilute constituents and the iron hydroxide floc.

The effects on survival times of the Edray strain of brook trout (*Salvelinus fontinalis*) at pH's 3.15 or 3.5 after a short exposure to a sublethal pH (pH's 5.0 or 5.8) were measured by Falk and Dunson,[64] but they observed that seasonal changes in resistance times were more marked than the effects of prior exposure to low pH. Heritable differences in acid tolerance of some inbred strains of brook trout exposed to pH 3.15 were also large; mean survival time for the most resistant strain was 16.8 hours compared to 6.3 hours for the least resistant strain. Trojnar[65] investigated the hatching response of brook trout eggs placed in gravel to incubation pH levels of 4.6 to 8.0 and observed the response of fry to a different pH (4.0 to 8.0) upon emergence. Survival of eggs to hatching ranged from 76% at pH 4.65 to 91% at pH 8.7, and fry incubated at pH 8.07 had 0% survival at pH 4.00 while fry incubated at pH levels between 4.65 and 5.64 had mean survival values between 60 and 76%. This suggested an acclimation effect, in that trout fry incubated at a lower, but sublethal, pH level are less likely to experience acid-induced mortality upon emergence than those incubated in spring upwellings. The

effect of low pH during incubation on the hatching success of white sucker eggs and on survival of sucker prolarvae to the swim-up stage in water of high and low conductivity was also evaluated by Trojnar.[66] In water of low conductivity (20 umhos/cm) no eggs survived to hatching at pH 4.16 and 4.48, whereas in high conductivity (250 umhos/cm) water a few prolarvae hatched at pH 4.5 but were deformed and soon died. At pH 5.0 and above the total hatch was the same at either conductivity level, but at 5.0 none of the prolarvae survived to swim-up due to deformities and abnormalities in the vascular system. At both conductivity levels there was a reduction in swim-up at pH 5.3 compared with the controls (pH 8.0 and 7.0), but none at pH 5.8. The results indicated that hardness had no influence on survival to hatching but did affect survival to swim-up, although a two-way analysis of variance revealed no significant interaction between pH and hardness. The effects of low pH on reproduction, growth, and survival of flagfish (*Jordanella floridae*) in soft water typically found in northern Ontario were described by Craig and Baksi.[67] Egg production, egg fertility, and fry growth were impaired at pH levels of 6.0, 5.5, 5.0, and 4.5 compared to that of controls held in untreated water (pH 6.8). Flagfish fry survival was reduced at pH 5.5 and 5.0, and no fry survived at pH 4.5. Adult mortality increased from 7 percent in controls to 64 percent in pH 4.5. The no effect level of pH depression for successful reproduction was determined to be pH 6.5. Ruby *et al.*[68] obtained similar results in a study designed to measure the reproductive capacity of breeding female flagfish exposed to different pH levels for 20 days. Compared to controls (pH 6.7) the ability of oocytes to form eggs was reduced to 20.7, 15.8, 2.1, and 8.2% at pH's 6.0, 5.5, 5.0, and 4.5, respectively, as shown by quantitative histological analysis of oocyte numbers in six arbitrarily assigned development stages. This reduction was attributed to failure of the oocytes to deposit secondary yolk within the cytoplasm at all pH levels below the control. At pH 4.5 both primary and secondary yolk deposition were strongly inhibited, and protein synthesis was also affected at all pH levels below 6.7. Based upon the reproductive indices calculated for oogenesis it was concluded that the pH should not fall below pH 6.0 for successful reproduction of this species. Five developmental stages of Atlantic salmon (*Salmo salar*) embryos and alevins, after incubation and fertilization at pH 6.6 to 6.9, were subjected by Daye and Garside[69] to several levels of low pH ranging from 2.7 to 4.3. Embryos in early cleavage were more sensitive to low pH than older encapsulated embryos, and alevins were more sensitive to low pH than embryos. The lower lethal limits were about pH 3.6, 3.0, and 4.0 for embryos during early cleavage, older embryos just before hatch, and alevins at 7 and 28 days after median hatch, respectively. Daye and Garside[70] reported on the histological effects suffered by fingerling brook trout acclimated at 15°C and pH 6.8 and subjected to acute and chronic pH stress for up to 10 000 minutes in a series of continuous-flow dilutions of sulfuric acid or sodium hydroxide from pH 2.2 to 10.8 at 20°C. No difference in the degree or form of tissue injury were detected between series for corresponding pH levels at 10 and 20°C, and thresholds for tissue and cellular derangements were pH 5.2 and 9.0. Progressive degrees of hypertrophy and excessive secretion of mucous occurred with increased pH stress to mucous cells of gills, nares, and integument. Epithelial necrosis and sloughing occurred extensively on gills, cornea, integument, and, at lethal levels (pH 3.5 and 9.8), in the esophagus.

Dively *et al.*[71] investigated the extent and duration of hypoxia and acidosis, and compared hemal response patterns occurring in summer- and winter-collected brook trout during early acclimation stages to sublethal acid toxication (pH 4.2). All acid exposed trout showed elevated P_{CO_2} hematocrit, and ventilation rates, along with signs of behavior distress and serum Na losses, but other than a transient drop in P_{O_2} June-exposed fish were neither hypoxemic nor was there evidence of blood acidosis. Summer-exposed trout actually showed a moderate alkalosis. Mudge *et al.*[72] determined the effects of low environmental pH on the histology and histochemistry of the adrenal (interrenal) tissue of brook trout. Trout exposed to near-toxic levels of acidity (pH 4.0) showed a reduction in interrenal cell size and decreased cytoplasmic RNA content (azure B-RNA decreased 27% over a 24-hour exposure), suggesting an inhibition of RNA synthesis, and presumably of steroidogenesis, leading to a depressed cellular metabolism. Additionally, mitotic analysis revealed these cells to be diploid and nonreplicating. Nor was there any loss in DNA content. Several biochemical properties of blood serum and other tissues from white suckers and brown bullheads (*Ictalurus nebulosus*) inhabiting an acid lake were measured by Lockhart and Lutz.[73] Despite the presence of deformities, examination of bone in white

suckers for N, Na, Mg, P, K, and Ca showed no demineralization. Values for hemoglobin failed to indicate an anticipated adaptive response to asphyxia, and normal values for hemoglobin, hematocrit, blood sugar, serum protein, and Ca content of scales showed that starvation was not the explanation for slow growth rate in suckers and bullheads. However, maturing females in highly acid Lake George failed to show expected elevated serum Ca relative to males, as did females from non-acid locations where maturation stages were similar, and it was suggested that Ca dynamics were altered in fish found in acid waters suffering reproductive failure.

PESTICIDE POLLUTANTS

Insecticides. The occurrence of pesticide residues from the water, stream bed material, fish, and soil collected in four small drainage basins in Pennsylvania was examined by Truhlar and Reed.[74] All water and fish samples contained pesticide concentrations less than the maximum recommended level. DDT or one of its metabolites was the most frequently occurring insecticide. The highest concentration in fish was 0.590 μg/g. Other insecticides such as dieldrin were found in the soil and water. A study was conducted by Paz[75] to investigate the presence of DDT in the Jordan watershed. DDT residues in fish averaged 0.37 μg/g in carp, 2.59 μg/g in benith, and 3.34 μg/g in sardines. DDT concentration increased with age and weight of the fish. The distribution of residual quantities of DDT in fish of the Kiev Reservoir, U. S. S. R. was studied by Polishchuk and Matvienko.[76] The content of DDT residues and its metabolites DDE and DDD was greatest in the visceral fat, somewhat less in the gonads and liver, and usually undetected in the muscle. Residue content was also affected by the weight and age of fish and was higher in predatory fish (pike, zander, perch) than in nonpredatory fish (roach, bream). Museum specimens of six species of Lake Michigan fish collected from 1929 through 1966 were analyzed for dieldrin, polychlorinated biphenyls (PCBs), and DDT analogs by Neidermyer and Hickey.[77] Low residues of dieldrin appeared first in 1955 and exhibited no trend with time. Both PCBs and DDT increased progressively through 1965 and were always detected together. DDT concentrations peaked in 1965. A study to determine the cause for the decline of fishing success in Sebago Lake in Cumberland County, Maine was conducted by DeRoche[78] of the Maine Department of Inland Fisheries and Wildlife. The results showed that the use of DDT to control insects was responsible for the collapse of the smelt and salmon populations. Since the cessation of the use of DDT, populations of the fishery are returning to normal. Residues of DDT and its metabolites were down to levels not considered harmful to fish life. The deposition of dieldrin in the water, soil, plants, and fish in flowing and non-flowing aquatic systems was examined by Lorio *et al.*[79] The distribution of dieldrin was similar in the waters of both systems. Soils contained little dieldrin but the compound was accumulated rapidly and as high as 0.474 and 3.95 μg/g by plants and fish, respectively. Concentrations of dieldrin in soil, plants, and fish declined with time. Burress *et al.*[80] conducted field tests to document the efficacy of isobornyl thiocyanoacetate (Thanite) as an aid in live fish collection. Twenty-six species were collected alive after 1.4 μl/l applications of Thanite. With the exception of northern pike (*Esox lucius*) most fish recovered quickly after being placed in fresh water. Most fish collected within 1.5 hours after treatment survived, but survival rate decreased with time of exposure. Endosulphan was found to be the cause of a sudden fish kill near the village of De Kwake, Netherlands (Kerkoff and Boer[81]). Concentrations of endosulphan in dead fish were as high as 72 μg/g. Biological effects and persistence of methyl parathion in Clear Lake, Calif. were examined by Apperson *et al.*[82] Residues in bluegill (*Lepomis macrochirus*) exposed to treatments varying from 0.50 to 5.40 μg/l ranged from 0.011 to 0.11 μg/g which corresponded to a 39- to 28-fold increase relative to water concentrations. A significant correlation was found between the methyl parathion levels in fish and water collected simultaneously. Plumb and Richburg[83] investigated a continuous mortality of channel catfish (*Ictalurus punctatus*) and brown bullheads (*Ictalurus nebulosus*) in two ponds in Alabama. Sera of dying catfish averaged 0.29 to 1.8 μg/g of endrin and 1.71 to 1.76 μg/g toxaphene during three sampling periods. Bullheads contained 2.40 μg/g of endrin and 0.53 μg/g DDT in the sera and 0.60 μg/g endrin in the brain. Sera from two healthy populations of catfish taken from the ponds the next year had less than 0.02 μg/g of endrin and 0.10 μg/g of toxaphene.

Davey *et al.*[84] studied the toxicity of five ricefield pesticides to the mosquitofish (*Gambusia affinis*) and green sunfish (*Lepomis cyanellus*) under laboratory and field conditions in Arkansas. In laboratory tests, green sunfish were more susceptible than mos-

quitofish to all pesticides except molinate. Parathion was the most toxic compound to both species and carbofuran the least. All pesticides with the exception of parathion after 72 hours and chlorpyrifos after 24, 48, and 72 hours killed less than 10% of all fish tested when applied at recommended field dosages. Studies of methoxychlor residues in caged and wild fish from the Athabasca River, Alta. after a single application of this compound were made by Lockhart *et al.*[85] Caged fish were not killed by a nominal concentration of 300 µg/l for 15 minutes and contained lower liver residues than wild fish of the same species captured near the cages. Residues in fish were below detection 12 days after treatment.

Dean *et al.*[86] determined the toxicity of methoxychlor and naled to different life stages of landlocked Atlantic salmon (*Salmo salar sebago*). For methoxychlor, the 24-hour LC50 to feeding fry and yearling was 92 and 75 µg/l, respectively, and the 24-hour LC50 to yearlings was 54 µg/l. The 24-hour LC50 for naled to feeding fry the fingerlings was 165 µg/l. Fabacher *et al.*[87] determined the toxicity of DEF (a cotton defoliant) and methyl parathion (boll weevil insecticide) singly and in combination to mosquitofish. Results showed that 0.5 mg DEF/l caused no mortality after 24 hours, 5.0 mg/l methyl parathion caused 8 percent mortality, and a combination of the two caused 89% mortality. It was concluded that this greater than additive toxicity effect should be considered when a combination of both are sprayed near aquatic environments. A rapid assessment study to determine the toxicity to and bioaccumulation by fathead minnow larvae and early juveniles of three chlorinated cyclodiene insecticide intermediates, hexachlorocyclopentadiene, hexachloronorbornadiene, and heptachloronorbornene, was conducted by Spehar *et al.*[88]; concentrations of 7.3, 122, and 83.5 µg/l of each compound, respectively, significantly decreased survival after 4 days. Growth of 30-day-old larvae was significantly reduced at 38.4 µg/l of hexachloronorbornadiene and 40 µg/l of heptachloronorbornene and was the most sensitive indicator of toxicity in each study. Bioaccumulation factors for fish exposed to less than 38.4 and 40 µg/l of each of these two compounds, respectively, were 6400 and 11 200. The acute and chronic toxicity of technical chlordane to fish and invertebrates was determined by Cardwell *et al.*[89] The 96-hour LC50 values for fish were: 36.9 µg/l for fathead minnows (*Pimephales promelas*), 47 µg/l for brook trout (*Salvelinus fontinalis*), and 59 µg/l for bluegills. In chronic tests, the lowest concentrations having adverse effects on fish were: 0.32 µg/l for brook trout and 1.22 µg/l for bluegills. The toxicity and accumulation of Imidan in freshwater invertebrates and fish was measured by Julian and Sanders.[90] Among fish tested, chinook salmon (*Orchorhynchus tshawytscha*) and small-mouth bass (*Micropterus dolomieui*) were the most sensitive (96-hour LC50, 150 µg/l) and channel catfish the most resistant (96-hour LC50, 11 000 µg/l). Toxicity increased with increased temperature and decreasing pH, but was unaffected by water hardness. Eyed eggs and yolk-sac fry of rainbow trout (*Salmo gairdneri*) were more resistant than fingerlings. Invertebrates and fish accumulated radioactive residues in 48 hours that ranged from 1 to 11 times the water concentration of 1.2 µg/l. Pillai *et al.*[91] studied the tolerance, uptake, and metabolism of DDT in the mosquitofish. At concentrations of 0.5 mg/l and above, the LT50 values were about 13 hours. The LC50 value for this species was 60 µg/l. Fish concentrated DDT by a factor of 10 in 24 hours with the maximum total DDT of 26.5 µg/g obtained after a 1-week exposure to 4 µg/l in the water. DDE was the major metabolite found in the fish. Jarvinen *et al.*[92,93] determined the chronic toxicity and accumulation of DDT in fathead minnows exposed to contaminated food and water. Higher total DDT residues were accumulated in fish from the water than the food with concentration factors of 100 000 and 1.2 from these sources, respectively. Toxicity residues from both sources were additive. Maximum acceptable toxicant concentrations for DDT were 0.9 µg/l for fish exposed to DDT in water only and 0.4 µg/l for fish exposed to DDT in water and food. DDT was eliminated from fish within 56 days. Loaches (*Misgurnus anguillicaudatus*) from Taiwan were treated with DDT, dieldrin, and monocrotophos by aqueous exposure and by injection by Yang and Sun.[94] DDT and dieldrin were 150 and 220 times more toxic, respectively, than monocrotophos to fish exposed in aqueous solutions but only ⅙ and ¼ as toxic as monocrotophos by injection after 24 hours. After 24 hours fish absorbed 96.5 percent of DDT, 92.7% of dieldrin, and 14.3% of monocrotophos from aqueous solution with the initial rates of absorption of DDT and dieldrin being 10 to 20 times faster than that for monocrotophos. The largest differences in relative toxicity of these compounds were thought to be due to partition distribution since DDT and dieldrin are lipophilic and monocrotophos is hydrophilic.

Jackson[96] reported the uptake and elimination of endrin in various tissues of channel catfish. After 147 days on an endrin-contaminated diet (2.5 μg/g, at 3% of the body weight) residue levels were 0.49 (whole body), 0.19 (liver), 1.53 (kidney), 1.09 (GI tract), and 0.63 μg/g (remaining carcass). After 44 days on a non-contaminated diet endrin was reduced to non-detectable levels in the kidney and liver and to 0.05, 0.04 and 0.03 μg/g in the GI tract, carcass, and whole fish, respectively. Shannon[98] measured the uptake and elimination of dieldrin by channel catfish. Fish exposed to 0.013 and 0.027 μg/l dieldrin reached equilibrium in 56 days. Fish exposed to 0.049 μg/l dieldrin did not reach equilibrium between uptake and elimination after 80 days. The accumulation of dieldrin increased with increased exposure concentration. Studies on the accumulation and metabolism of sumithion in rainbow trout and the southern topmouthed minnow (*Psudorusbora parva*) were made by Takimoto and Miyamoto.[97] Sumithion at 0.1 and 0.02 mg/l was readily accumulated by both species with bioaccumulation factors of 200–250 being reached after 1 to 3 days of exposure. Differences in the amount of accumulation varied with species, age, and water concentration. Sumithion decreased to 1/1000 during 5 days in fish transferred to clean water and was metabolized to several degradation products. The influence of lipid pool size on bioaccumulation of chlordane by northern redhorse suckers (*Moxostoma macrolepidotum*) was demonstrated by Roberts *et al.*[99] Tissue retention of chlordane in fish was directly proportional to the adiposity of the fish. For fish containing 2% lipid, the half-life of the *cis*-isomer was 60 days and for the *trans*-isomer, 33 days. The results indicated that the steady state concentration of chlordane in fish will not exceed the concentration of chlordane in the diet unless the adiposity of the fish is high.

Desaiah and Koch[99] determined the effects of several solvents on the inhibition of ATPase activities from catfish brain and cockroach muscle by DDT, toxaphene, and plictran. The results showed that inhibitory effects of toxaphene and DDT were greatly reduced by cyclohexane as compared to ethanol. However, ATPase inhibition by plictran showed no difference due to solvent. In general, the Na-K ATPase activity in catfish brain preparations showed less inhibition by DDT dissolved in non-polar solvents compared to ethanol than did Mg ATPase activities. Addison and Zinck[100] described the effect treatments of DDT, DDE, DDD, or DDMU on the subsequent dehydrochlorination of injected ^{14}C-DDT in brook trout fingerlings. The rate of dehydrochlorination of ^{14}C-DDT by trout was not influenced by these treatments and was more highly correlated with fish weight and lipid content. DDMU was cleared from the fish more rapidly than the other compounds. Jackson[101] investigated the effects of DDT on various membrane related functions in salmonid fish. When added *in vitro*, DDT inhibited both (Na + K)- and Mg-activated ATPases, but these effects did not correspond well with their generally observed *in vivo* toxicities. This inhibitory effect was an inverse function of temperature. Substrate competition studies and antagonism reactions were also investigated.

The *in vivo* effect of an organophosphorus insecticide, monocrotophos, individually and in combination with endrin and sevin were investigated by Thomas and Murthy.[102] Alkaline phosphatase activity in the liver, kidneys, and intestine was elevated by monocrotophos and its combinations with endrin and sevin. Catalase activity decreased in these treatments. Carbonic anhydrase activity was changed by treatment with several different combinations of the pesticides. Chambers[103] studied the relationship of esterases to organophosphorus insecticide tolerance in two populations of mosquitofish. A population highly resistant to organochlorine insecticides demonstrated a low tolerance to parathion and methyl parathion with the resistant population possessing higher levels of both peripheral acetylcholinesterase and carboxylesterases than the susceptible (unacclimated) population. Carboxylesterases, by their higher affinity for the organophosphates, may serve to protect acetylcholinesterase from inhibition.

The effect of 0.33 mg/l of parathion on general locomotor behavior of goldfish (*Carassius auratus*) was investigated by Rand.[104] The data demonstrated that parathion caused an overall decline in activity as measured by a decrease in the total number of entries into compartments, an increase in average duration of pathways, and a shift in magnitude to larger orientation angles leaving a compartment. A quantitative description of the effects of sublethal concentrations of DDT on the locomotor activity of bluegills was presented by Ellgaard *et al.*[105] DDT elicited hyperactive locomotor responses at all concentrations (0.008 to 0.2 μg/l) and the degree of such responses was concentration dependent. Maximum effects at each concentration were observed within the first 8 days of exposure.

The effects of DDT on locomotor activity were not reversed after fish were transferred to DDT-free water for 2 weeks. Wildish and Lister[106] studied dietary fenitrothion on growth and hierarchical position in brook trout. At a dietary level of 10 μg/g of wet food (11.5 μg/g dry food) fenitrothion did not effect growth or dominance patterns for trout at dry ration levels up to 4.45% of body weight per day. The method devised was thought to be useful in fish toxicology where effects of exposure to dietary contaminants must be determined for testing growth and food conversion efficiency, and in determining behavior dominance patterns.

Herbicides. Mann[107] evaluated the removal of weeds with herbicides in relation to its effect on fish. Aquatic plants were found to be not only carriers of fish prey species and cover for immature fish but also an important O_2 source in slow running waters. The significance of aquatic plants was discussed. The use of Cu as an aquatic herbicide was evaluated by the Maine Department of Environmental Protection.[108] It was recommended that Cu compounds for algae control be discontinued in Maine because they are toxic to various aquatic species including fish, nonselective and non-biodegradable. The chronic effects of Cu on aquatic animals were discussed. Sandhu[109] conducted a study to investigate the cause of a large fish kill in a reservoir near a cotton field following aerial herbicide spraying. Dead catfish along with some vegetation contained up to 5.09 to 7.35 mg As/l respectively, with As being the probable cause of the kill. Absorption of As was irreversible and its bioaccumulation continued as As levels in the water decreased. The effects of dipotassium endothall on rooted aquatics and adult and first generation bluegills (*Lepomis macrochirus*) in ponds in Delafield, Wisconsin, was investigated by Serns.[110] A treatment of 5.0 mg/l (active ingredient) endothall had no effect on the survival, growth, or reproduction of adult or first generation bluegills. Residue analysis showed that little endothall was incorporated into edible bluegill flesh after this treatment. The effects and persistence of endothall in ponds in Delafield, Wisconsin were determined by Holmberg and Lee.[111] The results showed that after a period of slow degradation (~12 days) endothall rapidly broke down reaching the level of detection (0.1 mg/l) in 18 days. The initial concentration of 5.0 mg/l endothall did not appear to cause any adverse effects on the survival of bluegills in the ponds. Lawson[112] quantified the amount of 2,4,5-T residues appearing in storm runoff from small upland watersheds in Arkansas as a result of spraying for vegetation control. None of the concentrations found in any of the streams were considered to be toxic to fish or other aquatic organisms.

The toxicity of diquat and cutrin to fingerling brown trout (*Salmo trutta*) was studied by Simonin and Skea.[113] The 96-hour LC50 values for these chemicals were 20.4 and 0.198 mg/l for diquat and cutrin, respectively. The data indicated the chemicals acted synergistically when in combination. The acute toxicity of atrazine on *Coregonus fera* fry under starvation conditions was investigated by Gunkel and Kausch.[114] The toxicity in relation to concentration as well as the effects of temperature and age were examined. McCorkle *et al.*[115] determined the acute toxicity of selected herbicides to fingerling channel catfish (*Ictalurus punctatus*). The 96-hour LC50 values for four herbicides found to be toxic were: propanil, 3896 μg/l; trifluralin, 417 μg/l; bensulide, 379 μg/l; and DNBP, 118 μg/l. The toxicity of Dyanap®, a mixture of DNBP and alanap, was less than that of DNBP. Rehwoldt *et al.*[116] investigated the acute and chronic effects of selected herbicides and pesticides on several species of freshwater fish. In acute studies toxicity varied with fish species. In all species 2,4-D and 2,4,5-T were the least toxic. Concentrations of 0.1 mg/l 2,4-D and 2,4,5-T and 0.01 mg/l malathion in chronic tests caused no observable effects on growth but substantially reduced acetylcholinesterase levels. A bibliography on the toxicity of the herbicides, acrolein, dalapon, dichlobenil, diquat, and endothall, to aquatic organisms was compiled by Folmar.[117] The data were listed in tables which included test organisms, type of test, experimental conditions and test results. Each table was followed by a list of references. Walsh *et al.*[118] determined the toxicity, rate of residue uptake, and elimination and potential effect of xylene for causing off-flavor in flesh of rainbow trout (*Salmo gairdneri*). The 96-hour LC50 value for xylene was 13.5 mg/l. The toxicity of xylene to trout in artificial streams closely approximated results obtained in laboratory studies. Continuous exposure of trout to xylene concentrations of 0.36, 0.74 and 1.3 mg/l for 56 days caused a significant off-flavor. GLC analysis indicated that both uptake and elimination of xylene in trout was rapid. Residues of simazine were analyzed from the mud, water, benthic invertebrates, and fish in ponds at the Fish-Pesticide Research Laboratory, near Columbia, Missouri,

by Mauck *et al.*[119] After treatment of 0.1 to 3.0 mg/l simazine, simazine was present in bluegills from all treated ponds up to 1 year after the initial application, but rapidly declined between 142 and 176 days after treatment. Residues in bluegills did not cause any observable adverse effect.

Piscicides and anesthetics. Chemical treatment with Fintrol (Antimycin A) for control of stunted panfish in Silver Lake, Columbia County was conducted by Congdon.[120] The objective was to re-establish a balanced fish population in the lake. A predictable kill was achieved by treating the lake in late fall with 1.5 μg/l antimycin. The importance of environmental factors and bioassay procedures on treatment was discussed. Bartoo[121] reported that between 15.3 and 20.3% of the yellow perch (*Perca flavescens*) population in North Lake, Washington surfaced within 96 hours after a rotenone treatment of 0.5 mg/l. It was determined that the proportion of a fish population surfacing within the first few days after a rotenone treatment drops rapidly with water temperature.

Paflitschek[122] examined the toxic effects of Bayer 73 on eggs and yolk-sac larvae of *Tilapia leucosticta*. Sensitivity increased with age, with yolk-sac larvae being more sensitive than the fertilized egg. LC50 values for 3-day-old eggs and yolk-sac larvae were 8.4–11.4 and 1.5 μg/l, respectively. In all experiments different degrees of deformity appeared in the course of development. The acute toxic effects of Bayer 73 and TFM on several species of invertebrates were determined by Rye and King.[123] Sea lamprey (*Petromyzon marinus*) larvae and fingerling rainbow trout (*Salmo gairdneri*) were included in the study for reference. The 24-hour LC50 values for sea lampreys and rainbow trout exposed to Bayer 73 were 0.049 and 0.14 mg/l and to TFM were 0.78 and 3.85 mg/l, respectively. Cairns *et al.*[124] analyzed the response of snails and brown trout (*Salmo trutta*) exposed to stress following acute sublethal exposure to TFM. Prior and concomitant exposure to 2 mg/l of TFM significantly reduced the median survival time for trout subsequently exposed to a lethal thermal shock. Marking and Bills[125] found that goldfish (*Carassius auratus*) and black bullheads (*Ictalurus melas*) were the most resistant of several species to Noxfish (5% rotenone) and Atlantic salmon (*Salmo salar*) was the most sensitive. Toxicity was influenced little by temperature, water hardness, or pH. In general, Noxfish was more toxic in static tests than flow-through tests. Potassium permanganate was an excellent detoxifier of Noxfish while Cl was less efficient. The toxicity of furanace to fish, aquatic invertebrates, and frog eggs and larvae was determined by Marking *et al.*[126] The 96-hour LC50s ranged from 0.820 mg/l for fathead minnows (*Pimephales promelas*) to 3.00 mg/l for bluegills (*Lepomis macrochirus*) and from 1.13 to 20 mg/l for six species of invertebrates. Toxicity increased with elevated temperatures in tests with rainbow trout, channel catfish (*Ictalurus punctatus*), and green sunfish (*Lepomis cyanellus*). Bills *et al.*[127] studied the acute toxicity (96-hour LC50) of malachite green on selected fishes and aquatic invertebrates. Bluegills were the most sensitive (0.0305 mg/l) and coho salmon (*Oncorhynchus kisutch*) the most resistant (0.383 mg/l). Invertebrates exposed were generally more resistant than fish. The toxicity of malachite green was generally not affected by water hardness or pH, but increased slightly with water temperature. Malachite green did not detoxify after 3 weeks, but was adsorbed readily by activated carbon. The effect of malachite green and formalin on the survival of largemouth bass (*Micropterus salmoides*) eggs and fry was studied by Wright.[128] The data indicated that formalin could safely be used as a fungus control for eggs at the standard concentration of 4000 mg/l for 10 minutes, and that fry could be safely treated with formalin at 50 mg/l for 60 minutes. Malachite green, however, decreased survival of eggs and larvae in all concentrations from that of the control. The acute toxicity of formalin to selected fishes and aquatic invertebrates was evaluated by Bills *et al.*[129] The black bullhead and channel catfish were most sensitive to formalin (96-hour LC50s were 62.1 and 65.8 μl/l, respectively) and Atlantic salmon and green sunfish were the most resistant (96-hour LC50 for each was 173 μl/l. Formalin was more toxic to some species in warm water than in cold water. Formalin solutions were not detoxified by oxidation or reduction or activated carbon and persisted after 3 weeks of aging.

Residue analysis of TFM in lake trout (*Salvelinus namaycush*) from Lake Superior and chinook salmon (*Oncorhynchus tshawytscha*) from Lake Huron was conducted by Sills and Allen.[130] No residues of TFM (limit of detection, <0.01 μg/g) were found in any of the samples (25 per species) indicating that the lampricide was not biomagnified. Allen and Hunn[131] studied the renal excretion of channel catfish following injection of quinaldine sulfate or TFM. Catfish injected with 1.0 mg of either compound eliminated most of the doses

by extra-renal routes. Patterns of renal excretion of Na, K, Ca, Mg, and Cl appeared to be associated with the stress of the urine collection technique rather than the elimination of either compound. Concentrations of the five ions were determined in urine, plasma, and gall bladder bile.

Hattingh[132] examined the effect of MS-222 on the microhaematocrit value of fish blood. Blood containing 0.08 mg/ml of MS-222 showed a higher haematocrit value than blood without the anesthetic. Treated blood was also haemolysed while blood without MS-222 was not. The effects of anesthesia with MS-222, neutralized MS-222 and benzocaine on the blood constituents of rainbow trout were evaluated by Soivoi *et al.*[133] After anesthesia for 15 minutes with 100 to 150 mg/l MS-222 and 33 mg/l benzocaine, Hct values, Hb concentrations, and blood lactate concentrations increased, with initial values being restored within 4 to 12 hours. All anesthetics raised Mg concentrations. Neutralized MS-222 and benzocaine elevated the plasma K concentrations more rapidly than unbuffered MS-222. Guerin-Ancey[134] conducted a study of the nitrogen excretion of bass (*Dicentrarchus labrax*) during growth and the effects of handling and MS-222 on the excretion of ammonia and urea. Ammonia and urea nitrogen excretion were found to increase with handling, which increased nitrogen metabolism. Handling had no effect on metabolism and excretion of MS-222 treated fish, but MS-222 slowed the metabolism of anesthetized fish. The influence of specimen weight upon induction and recovery from Stage 1 MS-222 was examined in goldfish, brook trout (*Salvelinus fontinalis*), and rainbow trout exposed to various concentration-temperature combinations by Houston *et al.*[135] Both induction and recovery times varied inversely with weight, the influence of weight being particularly pronounced among smaller specimens. Grizzle[136] examined the hematological changes in fasted and fed fingerling channel catfish exposed to 0.1 mg/l of malachite green. In both tests, neutrophilia at 1 and 3 days after treatment was the most drastic hematological change found. Other less significant changes occurred between 3 and 21 days of exposure, with no difference found after 28 days. The effects of malachite green on the blood chemistry, hematology, and histopathology of juvenile coho salmon were determined by Bills and Hunn.[137] A concentration of 0.1 mg/l of malachite green had no adverse effect on the blood, but resulted in an increase in K concentration in the plasma. Histopathological examination of the fish yielded no discernible effects except that the kidney and spleen of the controls contained more macrophages than those exposed. The effects of rotenone on blood respiration in green sunfish were studied by Perry and Conway.[138] The results indicated that 0.25 mg/l of rotenone caused toxicity because of reduced O_2 withdrawal from blood entering tissue capillary beds. The authors concluded that rotenone acts primarily by inhibiting O_2 utilization by fish cell mitrocondria.

CHEMICAL POLLUTANTS

Inorganics. Lewis[139] evaluated the influence of an open-pit copper mine of an upper sonoran intermittent stream ecosystem (Pinto Creek) in Arizona. The effects of suspended solids increase on the biota overshadowed the importance of all other quality criteria. Copper and Zn, singly or in combination, exceeded toxic levels to fish in 25 percent of the water quality samples. It was concluded that the Pinto Creek biota will be seriously jeopardized, if not destroyed, if present mining practices are continued. The validity of laboratory tests for predicting Cu toxicity in streams was evaluated by Geckler *et al.*[140] in Shayler Run, Clermont County, Ohio. Laboratory data were shown to be useable for the prediction of toxic effects in a natural stream during the 3-year stream exposure and, in general, was shown to be an underestimate of Cu toxicity because of the lack of observations of behavioral responses (principally avoidance) in the usual laboratory tests. Van Loon and Beamish[141] investigated the relationship between heavy metal contamination by atmospheric fallout and the effects on fish populations in several Manitoba lakes in the vicinity of a Cu, Zn, Ag, and gold sulfide ore mine and smelter. No adverse effects on fish populations were observed at concentrations of 90 μg Zn/l and 10 μg Cu/l; however, some long-term adverse effects did occur with some species when subjected to Zn concentrations between 200 and 1000 μg/l. Fish were more tolerant of these high Zn concentrations than was expected on the basis of laboratory toxicity tests. Södergren[142] studied the effects of heavy metal discharges of Co, Ni, Hg, Zn, Cu, and Fe on Atlantic salmon (*Salmo salar*) in a riverine ecosystem in northern Sweden. The 10-year study indicated that the ecosystem, as such, might be more sensitive to heavy metal pollution than are fish in laboratory experiments, and the possible direct toxic effects of Co on the fishes may have

been of lesser importance than the indirect effects via shortage of invertebrate fauna. As part of a survey of the limnology and fish populations of Red Indian Lake, Newfoundland, Canada, Morry and Cole[143] conducted a 96-hour, *in situ* bioassay using local brook trout (*Salvelinus fontinalis*) and landlocked Atlantic salmon obtained by electrofishing. No chemically-related mortality occurred during the test period. Copper and Zn levels ranged from 3 to 5 μg/l and from 50 to 328 μg/l, respectively. Bucke[144] described the incidence of tumors in a population of roach (*Rutilus rutilus*) in a lake fed by polluted streams. About 3% of the roach population was estimated to have tumors, principally on the pharyngeal region between the jaws. Pollutants in the lake included phenol, NH_3, Zn, Cu, Pb, and Cd, but no causal relationship with any of these was demonstrated. Kempf and Sittler[145] determined concentrations of Hg and organochlorine compounds in five species of fish (*Rutilus rutilus, Laciscus cephalus, Leuciscus leuciscus, Perca fluviatilis,* and *Esox lucius*) following a mass mortality of fish in the Rhine River. The high concentrations of these materials in the fish were thought to account for their increased mortality rate during 1973 through 1975. Possible causes of a massive fish kill in Lake Eyre, Central Australia were discussed by Ruello.[146] The fish, found in four parallel strands along the lake, consisted almost entirely of hairback herring (*Nematalosa erebi*) and hardyhead (*Craterocephalus eyresii*). Several likely causes of the fish kill were discussed, including low water temperature, an algae bloom followed by deoxygenation, and high salinity. Lake Eyre, which has a salt crust bottom and periodically dries and refloods with fresh water, has a one meter deep bottom layer with a salinity of greater than 300 percent, so it was thought likely that mixing of this water with the surface water by wind-induced seiches was the cause of the fish kills.

Birge and Black[147] examined the effect of borax and boric acid on the developmental stages of fish and amphibian embryos using a continuous-flow bioassay system. In general, B concentrations of 100 to 300 mg/l produced complete lethality for all species. No marked differences between boric acid and borax toxicity in hard and soft water were observed. As part of a study on the toxicity and uptake of B in the marine environment Thompson *et al.*[148] determined B (as sodium metaborate) toxicity to coho salmon (*Oncorhynchus kisutch*) alevins in freshwater. The 283-hour LC50 for freshwater coho alevins was 113 mg B/l. Tissue analysis for B residues was also performed on some of the freshwater coho that died during bioassay at the higher B concentrations. The toxicity of Cl to 12 species of freshwater fish was measured in soft reconstituted water by Marking and Bills.[149] Static 96-hour LC50s ranged from 0.156 mg/l for channel catfish (*Ictalurus punctatus*) to 1.41 mg/l for black bullheads (*Ictalurus melas*). Larson *et al.*[150] investigated the effects of changes in temperature, pH, and total alkalinity on the toxicity of inorganic chloramines to early life stages of coho salmon. The 96-hour LC50 varied according to life stage, the most sensitive stage being just after the fry stage. Acute toxicity was not affected by temperature and alkalinity but did increase at pH 8.1. Amiard[151] studied the toxicity of Co, Sb, Sr, and Ag to *Blennius pholis* and *Pleuronectes plantessa* and concluded that Ag was the most toxic, followed by Sb, Co, and Sr. The toxicities to 2-month-old rainbow trout (*Salmo gairdneri*) of nine metals found in drainage and process water from Au and Ag mines were measured by Hale[152] in a mobile bioassay laboratory using dilution water obtained upstream from the effluent drainage. Ninety-six hour LC50s were determined for As, Cd, Cr, Cu, Pb, Hg, Ni, Ag, and Zn. Cadmium, Ag, and Hg were the most toxic, in that order. Crawford and Allen[153] measured the toxicity of nitrite to chinook salmon fingerlings (*Oncorhynchus tshawytscha*) in both freshwater (32 mg Ca/l) and natural seawater (396 mg Ca/l, 32.5‰) by means of 48-hour static bioassays and also determined the percentage of hemoglobin oxidized to methemoglobin. The 48-hour median lethal nitrite concentration was 19 mg/l in freshwater compared to 10% mortality from exposure to 1070 mg nitrite/l for 48 hours. Further tests indicated that Ca inhibited nitrite toxicity, that possibly other ion(s) were involved, and that nitrite toxicity results from cause(s) other than methemoglobinemia.

Waiwood[154] determined the effects of Cu on mortality, critical swimming performance, growth rate, appetite, and hematocrit of rainbow trout as a function of pH and water hardness. The 240-hour LC20 for Cu ranged from 22 μg/l in low pH (6.0) and low hardness (30 mg/l) water to 300 μg/l in high pH (8.0) and high hardness (360 mg/l) water. The sublethal physiological responses that were measured were evaluated for their utility as criteria for predicting safe levels for Cu. The effects of various Cu ionic species on critical swimming performance and growth rate were discussed.

Zitko and Carson[155] found that the incipient lethal level (ILL) of Zn to juvenile Atlantic salmon varied from 150 to 1000 μg/l from fall through spring, with no detectable seasonal changes of water hardness, alkalinity, dissolved nitrogen, and humic acid concentration. The most sensitive period, as measured by the Zn ILL, coincided with the initial stages of the parr-smolt transformation and was, therefore, believed to be related to that transformation. Watson and McKeown[156] determined the effect of sublethal concentrations of Zn on rainbow trout. They concluded that growth was a useful parameter in assessing the toxicity of sublethal concentrations of Zn, especially when combined with plasma glucose measurements.

McIntosh and Bishop[157] determined the distribution of Cd, Zn, Pb, and Cr contaminants in a small eutrophic lake in northeastern Indiana and also used breathing rates and the cough response of bluegills (*Lepomis macrochirus*) to indicate sublethal heavy metal stress. Short-term bioassays with bluegills indicated no significant difference in Cd toxicity between fish from the contaminated lake and an uncontaminated lake. Birge *et al.*[158] determined the embryopathic effects of Cd, Hg, and Zn released from natural and metal-enriched systems as part of a study to develop egg culture bioassay procedures for monitoring bottom sediments for hazardous contaminants. Substantial frequencies of mortality and teratogenesis for goldfish (*Carassius auratus*) and rainbow trout when eggs were cultured over natural sediments enriched with as little as 0.1 to 1.0 mg Cd/l or Hg or 0 to 10 mg Zn/l. These results compared closely with independent ecological indicators of water quality, indicating that short-term egg hatchability tests may be used to predict long-term ecological effects of aquatic and sediment-bound contaminants. Effects of freshwater exposure to sublethal concentrations of Cu and Zn on the subsequent ability of yearling coho salmon to adapt to seawater and the effects of Cu on downstream migration of yearling coho salmon were investigated by Lorz and McPherson.[159] The percent survival in seawater was inversely proportional to the Cu concentration in freshwater, due to the loss of osmoregulatory activity; and Cu concentrations down to 5 μg/l, the lowest concentration tested, caused a reduction in the percentage of downstream migrants compared to controls when the coho yearlings were released into a natural stream. Sauter *et al.*[160] determined the effects of Pb, Cr, Cu, and Cd on eggs and fry of rainbow trout, lake trout (*Salvelinus namaycush*), brook trout, channel catfish, bluegill, white sucker (*Catostomus commersoni*), northern pike (*Esox lucius*), and walleye (*Stizostedion vitreum*) exposed for 60 days after hatching. The results were used to estimate a range of metal concentrations which bracket the maximum acceptable toxicant concentration (MATC). The effect of sublethal concentrations of Zn on the spawning potential of the zebrafish (*Brachydanio rerio*) was investigated by Speranza *et al.*[161] Both egg production and embryological development of the progeny were abnormal upon exposure to 5 mg Zn/l for 9 days. However, the Zn-exposed fish reproduced normally when returned to a Zn-free environment, indicating that brief exposure to Zn does not permanently alter the reproductive ability of the zebrafish. Sabodash[162] studied the effect of $ZnSO_4$ on the growth and development of progency from three different age groups of carp (*Cyprinus carpio*) females. The size of eggs and larvae were directly dependent on the age of the female carp. Zinc sulfate exposure of eggs resulted in premature hatching. Lind *et al.*[163] determined the long-term effects of HCN on the fathead minnow (*Pimephales promelas*) from hatch into the second generation juvenile period, with emphasis on long-term survival, growth rate, and reproductive success. The highest no-effect level was estimated to be between 12.9 and 19.6 μg/l, based on egg production. Smith and Sykora[164] studied the responses of brook trout and coho salmon eggs and alevins to laboratory exposures to lime-neutralized iron hydroxide suspensions. No measurable effect on hatchability, survival, and growth of brook trout alevins was observed up to 10.5 mg Fe/l; however, growth of 90-day-old coho salmon alevins was reduced in water containing 12.7 mg Fe/l, which led to the conclusion that the safe upper limit for hatchability, survival, and growth of coho salmon alevins was between 0.97 and 1.27 mg/l of lime-neutralized suspended Fe. Billard and de Montalembert[165] determined the toxicity to rainbow trout ova and eggs of iodophores added to the diluent for insemination or to freshwater. Freshly collected ova were sensitive to iodophores, with a fertility loss of about 10%. No fertilization occurred when iodophores were used during insemination, suggesting an unfavorable effect on spermatozoa. No significant loss of eggs was observed due to iodophores treatment after 30 minutes of water hardening.

The hypothesis that competition between

toxic cations and Ca and Mg ions for active sites in fish tissue was responsible for the influence of water hardness on lethality was investigated by Zitko and Carson.[166] Using time to 50% mortality as a measure of lethality to juvenile Atlantic salmon, it was determined that Zn toxicity was decreased by increases in both Ca and Mg, and that Cd and Cu toxicity were relatively unaffected. This behavior was explained by reference to the ratios of organic ligand binding constants for the survival cations. Perrone and Meade,[167] noting that coho salmon in the Rhode Island Agricultural Experiment Station were being maintained in apparently healthy condition at nitrite levels (up to 1.06 mg/l) in excess of those reported in the literature as being toxic to salmonids over extended periods, investigated the effect of water quality of nitrite toxicity. Results indicated that the tolerance of coho salmon to nitrite was increased when the chloride concentration was high. Shireman *et al.*[168] successfully treated columnaris disease in grass carp (*Ctenophryngodon idella*) by dipping the diseased fish in 500 mg $CuSO_4$/l for 2 minutes. Although all 12 fish eated shows signs of distress near the end of the treatment all survived both the disease and the treatment. Rødsaether *et al.*,[169] noting that the frequency of reported outbreaks of vibriosis (*Vibrio anguillarum*) has increased during the past few years in wild and in hatchery-reared fish, reported experiments demonstrating that eels (*Anguilla anguilla*) kept in Cu-contaminated (30 to 60 μg/l) fresh water die from vibriosis. As a working hypothesis it was proposed that a commensal relationship normally exists between fish and *V. anguillarum* and that this balance may be disturbed when the fish was poisoned or stressed. The effect of a naturally occurring stress (due to the presence of the cestode parasite (*Schistocephalus solidus*) on the response of the three-spined stickleback (*Gasterosteus aculeatus*) to the toxic action of Cd was investigated by Pascoe and Cram.[170] It was found that the parasitized sticklebacks had a significantly shorter median period of survival than non-parasitized fish. Survival times did not differ significantly between five Cd concentrations ranging from 0.5 to 5.0 mg/l but did differ significantly (5% level) between parasitized and nonparasitized fish. The 96-hour LC50 for non-parasitized sticklebacks was 6.5 mg Cd/l in a static test. Later, Pascoe and Mattey[171] extended the above study down to 0.001 mg Cd/l and also determined the amount of Cd present in the fish at death at each concentration. The static 96-hour LC50 in this study was 23 mg Cd/l, and death occurred at the lowest concentration studied in water of total hardness 103 to 111 mg/l. In all but three test fish the Cd concentration was found to be higher than the mean value for control fish and the concentration in parasites (*Schistocephalus solidus*) from four infected fish was less than 50% of that found in the fish tissue. Boyce and Yamada[172] investigated the difference in Zn susceptibility between juvenile sockeye salmon (*Oncorhynchus nerka*) infected with the intestinal cestode *Eubothrium salvelini* and non-infected smolts. When exposed to 1 mg dissolved Zn/l under laboratory conditions, infected smolts were significantly more susceptible to Zn than non-infected smolts.

Harden[173] determined As levels in largemouth bass (*Micropterus salmoides*), bream (*Lepomis* sp.), quillback suckers (*Carpiodes cyprinus*), carp, and brown trout (*Salmo trutta*) from Lake Hartwell, Lake Keowee, and Lake Jocassee in South Carolina. Measurable amounts of As were present in all fish samples; however, in only one case did any fish approach the FDA limit of 2.6 μg/g. Arsenic content had no apparent relation to Hg content. Davis[174] conducted a survey of Cd occurrence and concentration in fish from 11 major reservoirs in South Carolina. A total of 136 fish representing 13 species were collected and all fish contained less than 0.5 μg Cd/g with an average concentration of 0.138 μg/g. The reservoirs, therefore, do not presently present a hazard in terms of consumption of Cd with fish obtained from these lakes. Buhler *et al.*[175] investigated tissue accumulation and enzymatic effects of hexavalent Cr in 2-year-old rainbow trout reared in 25 μg/l and 2 to 10 μg/l. Highest concentrations observed were in the opercular bone, spleen, kidney, gastrointestinal tract, and gall bladder. Observed differences in enzyme activity between trout exposed to the two Cr levels were thought to be caused by factors other than Cr content of the water. Wright[176] measured the toxicity of fluoride in brown trout under static conditions. Dead animals taken from toxicity tests were used to determine a maximum tissue concentration factor of 2.3 in 5 mg fluoride/l after 160 hours. The uptake of Pb by goldfish was assessed by Merlini and Pozzi[177] using ^{203}Pb as a radiotracer. Only 8% of stable Pb remained in the ionic state when added (as $PbNO_3$) to the test water, and it appeared that Pb was accumulated in the ionic form. Merlini and Pozzi[178] also investigated the effect of

pH on the accumulation of Pb by the sunfish (*Lepomis gibbosus*). Fish kept in Lake Miggiore (Italy) water by pH 6.0 accumulated almost 3 times as much Pb as those kept at pH 7.5, indicating that accumulation was probably related to the concentration of Pb in solution. The initial major sites for Pb accumulation were the fins, gills, and liver. Muscle tissue contained the least amount of Pb after an 8-day exposure.

Total Hg concentrations in several internal organs and in the muscle tissue of white bass (*Morone chrysops*) and carp from Lake Erie were measured by Stiefel.[179] With few exceptions, the greatest accumulation of Hg in the white bass and carp occurred in the muscle. Relatively high concentrations in the kidneys, liver, and spleen, compared to the gills, led to the conclusion that the uptake of Hg for these fish was through the digestive system rather than through the gills. The kinetics of methyl mercury uptake in adult carp muscle, gill, pyloric caeca, and kidney was determined by Ekechukwu.[180] The gill accumulated the highest Hg concentration at the end of the 50-day exposure period, indicating that Hg penetration into the fish was probably essentially through this tissue. It was proposed also that the comparatively high Hg concentration in the pyloric caeca (second only to that in the gill) indicated that this tissue probably serves as an avenue of Hg elimination in carp. Braun[181] determined the relationship between Hg concentrations in water and concentrations in the gills and dorsal musculature of juvenile carp. Mercury concentrations in carp from unpolluted water, based on wet weight, was 0.09 μg/g in the dorsal musculature and 0.12 μg/g in the gills. At the highest water level studied (2 mg Hg/l) Hg content was 1046 μg/g in the gills and 6.49 μg/g in the muscle tissue. Mercury-adapted carp took up less Hg in acute toxicity tests than non-adapted carp. The ability of rainbow trout to methylate $HgCl_2$ administered through the gills or by ingestion with food was investigated by Pennacchioni *et al.*[182] The experiments showed that while $HgCl_2$ administered by either method increased the total Hg content of kidney, liver, intestine, and muscle tissue, no evidence was found to support the hypothesis that $HgCl_2$ was methylated in fish to form methylmercury. Lloyd *et al.*[183] measured Hg levels in the liver and muscle tissue of rainbow trout and brown trout from trout parks and streams of southern Missouri. Mercury levels were between 0.1 and 0.3 μg/g with no variation according to size and only slightly higher levels were found in the liver than in the flesh. These levels were substantially lower than those in fish taken 25 to 30 years ago from the same area, and the decrease was believed to be due either to a substantial decrease in Hg levels in the streams or to different stocking procedures now in effect. Giblin and Massaro[184] investigated the comparative uptake of methylmercury and methylmercury cysteine by rainbow trout kidney and red blood cells. The kidney preferentially took up methylmercury cysteine while the red blood cells preferentially took up unliganded methylmercury, suggesting that at least a portion of total renal Hg uptake is mediated via amino acid transport mechanisms. Steinnes *et al.*[185] measured the Hg content of perch (*Perca fluviatilis*) and brown trout downstream from a wood pulp factory during the years 1970 to 1975, following a ban on phenylmercury in the Norwegian pulp and paper industries beginning January 1, 1970. Muscle concentrations declined over a period of approximately 3 years to about 1 μg/g. In fish exposed to high Hg levels organ distribution studied indicated that very high Hg levels may be observed in the liver, kidneys, and possibly other organs while the muscular concentration is still at a moderate level. Only in cases of low exposure (muscular concentrations less than 0.5 μg/g) did muscle tissue concentration exceed that in most other tissues. Schindler and Alberts[186] compared the fate and possible transformation of Hg, Cd, and Cr in freshwater sediment-water environments, and as part of that study analyzed the mechanism of uptake and release of Hg forms by the mosquitofish (*Gambusia affinis*). It was concluded that physical adsorption processes rather than lipid solubility differences largely determined the initial rate of uptake. No evidence was found for methylation of Hg in *Gambusia*. Desorption rates of Hg from bed sediments and Hg transfer from bed sediments to guppies (*Poecilia reticulata*) and from fish to clean water were measured radiologically by Kudo.[187] No attempt was made to define the route of Hg from bed sediments into fish.

Kania *et al.*,[188] in a study of the fate and biological effects of Hg in artificial stream communities, measured the accumulation of Hg in mosquitofish at several levels of exposure. Fish exposed to 1.0 μg Hg/l over a 10-month period reached equilibrium levels of 4 to 7 μg/g. Olsson and Jensen[189] discussed the advantages of choosing a standard test organism for studying pollution patterns and enumerated several qualities which make pike a particularly advantageous test organism. Mer-

cury, DDT, and PCB levels in pike were determined, and for Hg all levels were above 0.4 μg/g in the Stockholm archipelago study area. In order to compare Hg levels in different regions it was necessary to consider the weight of individual fishes and to use the Hg level at a calculated standard weight. For DDT and PCB the mean levels in the fish samples could be used. As part of a larger study of the biochemical transformation and detoxification of Hg in the aquatic environment, Hamdy[190] determined the uptake of ^{203}Hg by guppies fed labeled mosquito larvae and by cichlids fed labeled guppies. Kim *et al.*[191] found that SeO_2 addition to $HgCl_2$ solutions resulted in increased whole body accumulation of Hg over $HgCl_2$ alone at low Hg concentrations (0.01 to 0.07 mg/l) and decreased accumulation in survivors at higher lethal Hg concentrations (0.10 to 0.16 mg/l). Spinelli and Mahnken,[192] based on reports that dogfish (*Squalus acanthias*) contained moderate-to-high amounts of Hg, attempted to define the limits to which dogfish meal can be included in diets used to rear pan-sized coho salmon. Results indicated that dogfish meal could be used as a replacement for up to 50% of the fish meal portion of the diet without encountering muscle Hg levels in excess of 0.5 μg/g. Abernathy and Cumbie[193] reported observations on Hg accumulation in largemouth bass from Lakes Hartwell, Keowee, and Jocassee in western South Carolina. Mercury levels were higher in fish from the latter two reservoirs, which were younger and relatively oligotrophic compared to levels in fish from the older and more eutrophic Lake Hartwell. The source of Hg in these reservoirs appeared to be the soil which formed their original sediments. Mercury accumulation data for largemouth bass and brown trout in Lake Jocasse, S. C. were analyzed statistically by Lienert and Rowell.[194] Statistical models were developed correlating fish length with Hg concentration. Because yearly models for largemouth bass fit the data but the pooled model for all years did not, it was concluded that there was a significant decrease in fish Hg concentration between 1974 and 1975, followed by a slight increase in 1976. The decrease was attributed to fish growth and dilution of accumulated Hg, and several possible explanations for the subsequent increase in concentration were discussed. None of the models developed using brown trout data were acceptable at the 95% confidence level. Koli *et al.*[195] analyzed fish samples consisting of nine species from rivers, lakes, and ponds throughout the state of South Carolina and found that in those species for which wide ranges of weights were analyzed, the larger fish had the highest Hg levels. Mercury levels exceeding the FDA guideline of 0.5 μg/g were found in mudfish from the Edisto River and in pike from Lake Morray.

Miller[196] exposed bluegill sunfish to ^{54}Mn-contaminated water at 10, 20, and 30°C; 4, 8, and 16 mg/DO/l; and for 6, 24, 96, and 168 hours. These exposures showed ^{54}Mn uptake patterns moderately affected by temperature, only negligibly affected by DO, and strongly affected by sampling time. After transfer to ^{54}Mn-free water, a short, rapid initial release of ^{54}Mn was observed followed by a long slow release phase. Largemouth bass in clean water which were fed prey previously exposed to ^{54}Mn-contaminated water showed less ^{54}Mn uptake 3 days after feeding than largemouth bass held in ^{54}Mn-contaminated water. Adams[197] measured Se concentrations by spectrophotometric procedures in water, sediment, zooplankton, and fish from the western basin of Lake Erie and determined the acute toxicity of sodium selenate and selenite in static and flow-through tests. The average concentration of Se (dry weight) in 10 fish species ranged from 8.12 ± 1.02 μg/g for sheepshead (*Archosargus probatocephalus*) to 1.80 ± 0.12 μg/g for common shiners (*Notropis cornutus*). Sodium selenite was found to be more toxic than sodium selenate, and both compounds were more toxic in the flow-through than in the static tests. Sims and Presley[198] determined heavy metal concentrations in fish and other organisms from an actively dredged Texas bay. The fish (including several euryhaline species) appeared to be lower in Cd, Cu, Pb, and Zn than the same species from non-dredged and relatively unpolluted areas. The liver tended to concentrate Cd, Cu, and Zn, while the heart concentrated Pb. Lowest levels of metals were found in the stomach and flesh. Brown and Chow[199] determined concentrations of Cd, Cu, Pb, Zn, and Hg in 15 species of fish (alewife, brown bullhead, carp, freshwater drum, gizzard shad, golden shiner, lake whitefish, largemouth bass, longnose sucker, pumpkinseed, rainbow smelt, rock bass, white bass, white sucker, and yellow perch) from Baie du Doré, Lake Huron, and Toronto Harbor, Lake Ontario. The concentrations of metals were similar in the various species studied and ranged in ascending order of relative abundance in muscle tissue from Cd, Hg, Pb, and Cu, to Zn. In a study designed to examine the feasibility

of eel culture in power station cooling water, Romeril and Davis[200] measured the growth rate and metal accumulation levels in the European eel in condenser water, cooling pond water, and in river water. It did not appear that eels cultured at elevated temperatures in the power station cooling water accumulated abnormal amounts of Cd, Cu, Fe, Ni, or Zn. Kent[201] analyzed the accumulation and distribution of organochlorine compounds and heavy metals in American Falls Reservoir, Idaho. Mercury and Cd were found in all fish species analyzed. Arsenic was found only in the sediments and water. Chlorinated hydrocarbon residues (DDT metabolites, dieldrin, and PCBs) were found in fish flesh but not in water samples. Brooks *et al.*[202] determined Hg, Cd, Cu, Fe, Mn, and Zn levels in various organs of rainbow trout and brown trout from lakes and rivers in central North Island, New Zealand. Mercury in the flesh of fish from several lakes averaged substantially above the recommended dietary maximum of 0.5 μg/g; however, none of the other metals were present in edible flesh in concentrations likely to cause a public health problem. Mercury was assumed to have been derived in part from a pulp and paper mill in one lake and from adjacent geothermal areas in other localities. The distribution of Cd and Pb in a rural, non-industrial-use stream ecosystem was analyzed by Enk and Mathis.[203] In general, concentrations of both metals increased successively from water to fish to sediments to aquatic invertebrates. Nabrzyski[204] determined Hg, Cu, and Zn content in muscle tissue of *Blicca bjoerkna, Abramis brama, Esox lucius, Tinca tinca, Salmo salar, Vimba vimba,* and *Perca fluviatilis.* Mercury content of fish in the Lower Vistula River (Poland) was high in the spring of 1972, but declined with time. Copper and Zn contents were no higher than natural levels. Terharr *et al.*[205] described a dynamic laboratory aquatic ecosystem designed to be capable of simultaneously distinguishing between bioaccumulation from the surrounding environment by all processes and biomagnification through successive trophic levels. Mercury and Ag were chosen to test the system because of their known biological differences with respect to accumulation. Organisms used in the system were algae, *Daphnia magna,* freshwater mussels, and the fathead minnow. Matthiessen and Brafield[206] studied the uptake and loss of sticklebacks of both stable Zn and ^{65} Zn in hard and soft water for periods of up to 400 hours in order to investigate the protective effect of Ca on Zn toxicity and to clarify the mechanisms of Zn uptake and loss. Sticklebacks were found to be able, after the initial Zn influx, to reduce their internal Zn levels to near-normal despite continued exposure to 1 and 4 mg Zn/l, an ability shown by no other fish species so far tested. The return to near-normal internal Zn concentration was believed to be due to the combined effects of relatively low Zn uptake and a Zn excretion system. Possible mechanisms and supporting evidence for this hypothesis were discussed.

In order to determine if interference with Zn metabolism by Cd could be correlated with the modifications of Zn metalloenzymes, Piavaux[207] exposed sunfish to several concentrations of Cd. Alkaline phosphatase activity was determined in various organs, along with Cd and Zn concentrations. Although Cd accumulated in all organs (particularly liver and kidneys) of treated fish, enzyme activity did not follow any systematic trend. Koyama and Itazawa[208] analyzed the effects of oral administration of Cd on the blood and bone chemistry of carp. Alkaline phosphatase activity and Ca content of serum, the Ca content of vertebrae, and cranial ash content decreased with increasing Cd content in the food while serum inorganic phosphorus concentrations increased. Violent swimming and tetany in some of the fish was thought to be due to hyperexcitability of the nervous system and muscles resulting from the low Ca and high inorganic phosphorus in the plasma. Cadmium concentrations in the food ranged from 0 to 5700 μg/g dry weight. In a companion paper, Koyama and Itazawa[209] discussed the morphological changes induced in the same carp. Abnormal black granules accumulated around blood vessels and in the liver, and in the kidney degeneration of proximal renal tubules and loss of protoplasm from cells were observed. This generation of the renal tubules was proposed as the initial event in a sequence leading through disturbance of the balance between Ca and inorganic P in the plasma, low Ca content of the vertebrae, hyperexcitability of the nervous system and muscles, violent swimming, and tetany to lateral curvature of the vertebral column. The utility of delta-amino levulinic acid dehydratase (ALA-D) activity in fish as a short-term indicator of harmful exposure to Pb was evaluated by Hodson *et al.*[210] Exposure of rainbow trout, brook trout, goldfish, and pumpkinseed (*Lepomis gibbosus*) to Pb consistently inhibited ALA-D within 2 weeks at concentrations which, for rainbow and brook trout, were close to published minimum

sublethal effect levels. Since near lethal exposures to Cd, Cu, Zn, and Hg showed no significant ALA-D inhibition it was concluded that ALA-D activity provides a fast, consistent, specific, and sensitive estimate of Pb levels causing sublethal harm to fish. Kendall[211] described the sequential toxic effects in catfish liver produced by an acute (LD50) dose of methylmercuric chloride. After 24 and 48 hours the livers appeared similar to controls upon gross inspection, but were noticeably different at 72 and 96 hours. Histological alterations are described beginning at 24 hours after interperitoneal injection. The effects of a Zn stress on the biochemical and physiological functions of fathead minnow cell cultures were described by Adragna.[212] Cell volume increased with increased Zn in the medium, and protein synthesis was accelerated. The activity of a number of purported Zn metalloenzymes were investigated, including lactate and malate dehydrogenase, and alkaline phosphatase. The cell cultures were able to adjust to a Zn stress if the increase was gradual, rather than abrupt. Zeitoun *et al.*[213] examined the contribution of essential mineral elements to developing rainbow trout eggs, embryos, and larvae from the water environment and from the unfertilized oocyte. Over the entire study period, at least 70% of the Ca, 76% of the Na, 45% of the K, 68% of the Fe, and 2% of the Zn in the larvae were derived from the water environment. All of the P and Cu could have been derived from the unfertilized oocyte. The relative roles of environmental Ca and the pituitary on water and electrolyte regulation in brown trout were investigated by Oduleye.[214] Increasing Ca concentrations resulted in a decrease in plasma electrolyte level and total electrolyte excretion. However, for hypophysectomiated fish, Ca was effectual in partially preventing the drop in plasma electrolytes resulting from hypophysectomy. A biological multichannel analyzer to measure mixed cell populations by cell size was developed by Curby *et al.*,[215] using peripheral whole blood from the euryhaline fish *Fundulus heteroclitus*. In the development of the instrument, alterations in cell size and frequency distribution in response to changes in salinity and exposure to Cu were measured.

Organics. Drapeau[216] discussed how polychlorinated biphenyls (PCB), detected in the environment since 1966, threaten the survival of several species of fish and ichthyophagous birds, and how they are capable of altering the equilibrium of aquatic ecosystems and harmfully affecting the health of man. Walker[217] summarized results of the Freshwater Fish Program which began monitoring fish samples for PCBs in 1970, reviewed several studies of chronic and acute toxicity of PCBs to aquatic organisms, and discussed potential abatement methods for controlling pollution due to PCBs. The presence and extent of contamination of water, sediments, and biota of the Hudson River by industrial use and discharge of PCBs were described by Nadeau and Davis.[218] Sediment and water samples collected immediately below a hydroelectric dam and power generating station contained 6 700 mg/gm and 2 800 μg/l Arochlor 1076, respectively. Whereas yellow perch (*Perca flavescens*) collected above the dam contained 17.0 μg/g Arochlors 1248 and 1254, Arochlors 1016 and 1242, or a mixture of the two, were found at levels of 350 μg/g in rock bass (*Ambloplites rupestris*) and 78.0 μg/g in common shiners (*Notropis cornutus*) collected at stations below the dam. At all collecting station the snail populations were important accumulators and concentrators of PCBs, with a tissue/water concentration factor as high as 45 000. Kaiser[219] identified organic contaminents and measured their background levels in three species of fish caught at various distances from a kraft and pulp mill effluent discharge in Nipigon Bay, Lake Superior. White sucker (*Catostomus commersoni*), longnose sucker (*Catostomus catostomus*), and yellow perch from all sites contained residues of PCBs and DDE at levels ranging from 0.04 to 0.80 μg PCB/g and 0.025 μg DDT/g on a wet weight basis. Most samples were also contaminated with hexachlorobenzene and several contained phthalic acid esters; and dehydroabietic acid, a known constituent of kraft mill effluent, was observed in one extract of each of the three fish species. Lake Superior lake trout (*Salvelinus namaycush*) had statistically higher levels of PCBs and DDT and its analogs than lake trout reared at a fish hatchery located within the watershed for Lake Superior and analyzed for chlorohydrocarbon residues by Parejko and Wu[220]. Hatchery confined lake trout contained mean PCB levels (Arochlors 1248, 1254, and 1260 combined) of 0.88 μg/g and a mean total of 0.68 μg DDT/g and analogs. Also detected in these fish samples were heptachlor and its analogs, delta-hexachlorocyclohexane (mean concentration of 70 μg/g) and a methoxychlor derivative. Because most of these compounds were detected in the standard diet of these fish, it appeared consistent that the lake trout consumed and absorbed chlorohydrocar-

bons from their diet. Miles[221] reported on chemical and pesticide levels in channel catfish (*Ictalurus punctatus*) upon re-opening of the Ohio River commercial fishery in 1973. Preliminary analyses indicated that the fishery, closed since 1970 due to excessive Hg levels in fish, faced possible problems due to high PCB levels. PCB levels were above the Food and Drug Administration's edible fish tolerance of 5.0 μg/g in channel catfish taken from one of three sampling locations, and, in addition, chlordane levels were above the acceptable limit of 0.3 μg/g at all three locations.

Water samples taken from a pristine, wilderness lake in Ontario were analyzed by Starnes and Brown[222] for the possible presence of biosynthesized, complex hydrocarbon mixtures. Total hydrocarbon content was 1.6 μg/l, with saturates accounting for 90% of the hydrocarbons present, and it was concluded that these were of natural origin. Masnik *et al.*[223] described the effects of a 13 000 l No. 2 fuel oil spill on the macroinvertebrates and fish in a small southwestern Virginia creek. Immediate mortality of water snakes and waterfowl was relatively high, but collection made at eight stations over a 5-month period indicated minimal mortality of benthos and fishes. Volatile chemical compounds present in Great Lakes salmon and environmentally related materials were identified by Lindsay.[224] Compounds that appeared to contribute to the intensely off-flavored salmon obtained from Lake Michigan included 1-octen-3-one, chlorbenzaldehyde and naphthalene, geosmin, methoxythiophenol, 2-, 3-, and 4-isopropyl phenols, and t-butyl phenol. Except for 1-octen-3-one, which occurs naturally in salmon, and geosmin, which occurs naturally in the environment, the compounds were probably derived from industrial pollution sources. The occurrence of a number of aromatic hydrocarbons along with substantial amounts of even-carbon numbered aliphatic hydrocarbons, themselves not odorous to any extent, suggested accumulation of oil and petroleum-based product contamination materials. Tabachek and Yurkowski[225] isolated microorganisms from saline lakes in southwestern Manitoba and identified those that produced the extracellular metabolites, geosmin and) 2-methylisoborneol, responsible for the muddy flavor found in fish and water in some lakes. Of 11 axenic or unialgal cultures of blue-green algae, 10 produced geosmin and one produced 2-methylisoborneol. Concentrations of the chelating agent ethylendiaminetetraacetic acid (EDTH) found in detergents, primary and secondary wastewater, and river water were determined by Gardiner.[226] Wastewater containing industrial effluents had concentrations >170 μg EDTH/l, and it was suggested that the presence of synthetic chelating agents may explain the survival of fish in water containing concentrations of trace metal, which in organically unpolluted water, would be considered lethal. Jensen[227] briefly reviewed the biocidal use, toxicity to aquatic species and mammals, persistence in water, and the bioaccumulation of organotin compounds. Included in the summarized toxicity data were LC50 levels in the range of 25 to 50 μg/l for several fish species. It was concluded with a survey of pollution abatement methods for these compounds. Hickey and Krenkel,[228] Yount,[229] and Brown[230] reviewed and discussed research efforts by the Tennessee Valley Authority, the Environmental Protection Agency, and the U. S. Department of Agriculture, respectively, directed toward assessment of the impacts that energy related activities may have on the aquatic ecosystem. Projected research included monitoring ecological recovery after reclamation of toxic spoils left by coal surface mining (TVA), the effects on aquatic bioata exposed to particulate and leachate from fine particulate coal while in transport and storage (EPA), and to determine the immediate and long-term effects and biological fate of energy related pollutants on freshwater ecosystems due to strip-mining (USDA).

Łuczak *et al.*[231] exposed *Daphnia magna*, the snail *Planorbis corneus*, and the guppy (*Poecilia reticulata*) to a range of chlorophen (technical polychlorbiphenyl) water concentrations from 0.1 to 100 mg/dm^3 (mg/l), and concluded that *D. magna* is the most sensitive animal which can be used for testing water contaminated with PCBs. Prior exposure of rainbow trout (*Salmo gairdneri*) to the PCB, Aroclor 1254, at levels of 0.01 and 0.1 μg/l significantly increased the toxicity of cyanide and Cr in tests conducted by Bills *et al.*,[232] while the toxicities of nitrate, nitrite, Cl, and Hg were unaffected by previous exposure to either concentration of the PCB. The 96-hour LC50s for cyanide to unexposed control fish and those exposed to low and high levels of PCB were 90.0, 66.2, and 65.8 μg/l, respectively, and those for Cr were 11.2, 9.0, and 7.05 mg/l, respectively. Klein *et al.*[233] reported on the toxicity to fish and attached periphyton growths (aufwuchs) of the jet fuels JP4 (a complex mixture of aliphatic and aromatic hydrocarbons) and JP9, and the

JP9 components RJ4 (isomers of tetrahydromethylcyclopentadiene dimer), RJ5 (isomers of tetrahydro-norbornadiene dimer), and MCH (methylcyclohexane). The 96-hour LC50 values for golden shiner (*Notemigonus chrysoleucas*) exposed to renewable static bioassays ranged from 0.5 to 70 µl/l of water emulsions of the fuels and components, 100 to 4700 µl/l of water in contact with pools of floating and sunken fuel, and for water soluble components of MCH and JP4, 23 500 and 20 000 µl/l, respectively. The water soluble components of RJ5 had the most effect on flagfish (*Jordanella floridae*) egg hatchability in continuous-flow bioassays, reducing hatchability at aqueous concentrations greater than 0.05 mg/l. In continuous-flow bioassays using the rainbow trout, 97-day LC50 values for the water soluble components of RJ4 and RJ5 were 0.045 and 0.072 mg/l, respectively, and the 23-day LC50s for MCH and JP9 were 1.3 and 0.38 mg/l, respectively. Both rainbow trout and flagfish accumulated MCH to 150x the water concentration, and trout concentrated RJ5 9800x and RJ5 3800x. The adaptability of *Huso huso* and *Acipenser stellatus* to elevated temperatures and their resistance to oil poisoning under thermal load conditions were studied by Velikhanov.[234] There was 100 percent mortality of great sturgeon at 30.3°C while at 21.9 to 27.2°C the mortality was nearly 9 times lower, and a petroleum concentration of 75 ml/l was lethal to 13-day-old great sturgeon. The results also revealed a pronounced effect of oil and thermal pollution on the O_2 regime; mass mortality of young stellate sturgeon in oil polluted water occurred upon a 6°C increase in water temperature, and 47-day-old great sturgeon did not survive a temperature increase as low as 3°C. A two-phase study by Matthews and Myers[235] established the acute toxicity of oil refinery wastewaters to redear sunfish (*Lepomis microlophus*). Phase I evaluated and compared toxicity of in-plant wastewaters of 17 oil refineries, compared toxicity results with chemical analyses of the samples, checked for presence of unknown toxic compounds, and catalogued behavioral activities of fish exposed to these wastewaters. The 24-hour LC50 values for redear sunfish exposed to these wastes ranged from 0.04 to >18% (by volume), but nine refineries had no samples which showed toxicity at the dilutions tested. Ammonia, sulfides, and phenolics, alone or in combination, were the major contributors to toxicity, and in some cases unknown hydrocarbons. Phase II samples, taken from various treatment stages of five oil refineries, showed decreasing toxicity with increasing treatment. Samples collected from the first treatment stage, gravity oil separate ion had 24-hour acute toxicity values of 4 to 50%, while succeeding activated sludge treatment reduced toxicity, except for those effluents containing high levels of ammonia. Samples taken from the final clarifier stage showed no toxicity for four of the five refineries tested.

Dolan and Hendricks[236] determined the toxicity of intact and biodegraded linear alkylbenzene sulfonate (LAS) surfactant to bluegill (*Lepomis macrochirus*) and the snail, *Goniobasis* sp. Acute toxicity to bluegills did not occur at 92% degraded surfactant, but 96-hour LC50 values ranged from 1.64 to 0.72 mg/l for 76.0 to 0% degraded LAS. GC analyses indicated that the homolog-isomer distribution of the LAS did not shift significantly until degradation was approximately 92% complete as indicated by methylene blue active substances measurements. Partial biodegradation of LAS was shown by Kimerle and Swisher[237] to significantly reduce the specific toxicity of the remaining LAS to *Daphnia magna* and the fathead minnow (*Pimephales promelas*). The 48-hour LC50 values for fathead minnows were approximately 10 000 mg/l for late biodegradation intermediates compared to 48-hour LC50 values of 5.3 to 86.1 mg/l for nonlinear LAS components, 0.4 to 43.0 mg/l for the different individual LAS homologs, and 1.7 mg/l for commercial LAS. The longer homologs and more terminal isomers were the most toxic, but were also degraded more rapidly under bacterial action. The results also emphasized that analysis for methylene blue active substances gave no basis for predicting the aquatic toxicity of an environmental sample because some of the biodegradation intermediates showed methylene blue activity but no significant toxicity. Oba *et al.*[238] determined and compared the effects of LAS and the biodegradtion products of LAS on mortality and hatching success of the Japanese killifish (*Oryzias laptipes*). The longer alkyl chain homologs and external phenyl isomers, although more toxic to fish, were more rapidly biodegraded, and it was shown that biodegraded LAS in a continuous-flow river model was less toxic for every stage of killifish than was intact LAS in a static bioassay system. It was concluded that the use of river models was a promising technique for evaluating the effects on aquatic organisms of easily biodegradable chemicals, such as LAS, present in the natural aquatic environment.

The lethality of differing phenol concentrations to goldfish (*Carassius auratus*) and carp (*Cyprinus carpio*) was investigated by Marinescu *et al.*[239] The results showed that carp were more sensitive than goldfish and that inanition strongly modified their resistance to the toxin. The influence of phenol on O_2 consumption in goldfish was also evaluated and the authors pointed out that O_2 consumption was a useful index in relation to phenol concentration and adaptation temperature. Fogels and Sprague[240] compared the short-term tolerance of zebrafish (*Brachydanio rerio*) and flagfish with that of rainbow trout exposed to five selected poisons and evaluated the two exotics as potential standard species for aquatic bioassays. Threshold or 10-day LC50s for the three species exposed to Cu, sodium pentachlorophenate (NaPCP), phenol, picloram, and dodecyl sodium sulfate (DSS) showed that, on the average, zebrafish were 2.6x and flagfish 4.2x as tolerant and trout, with the ratios of zebrafish/trout LC50s being considerably less variable than flagfish/trout LC50s. Responses of the two exotic species were not sufficiently different from those of rainbow trout, considering the known variable response of rainbow trout to a given toxicant in similar waters, to discount either species from consideration as a standard test-fish. In addition, phenol, NaPCP, and DSS were evaluated as possible reference toxicants, but only phenol met all the criteria considered. Miller and Mumma[241] compared the relative anesthetic activity and relative toxicity of various defensive steroids and norsesquiterpenes produced by water beetles from the families Dytiscidae and Gyrinidae and a wide selection of steroid standards to the fathead minnow. The 4-pregnen-3-ones and related derivatives, major components of dytiscid secretions, had LC50 values between 1 to 3 μg/l and were among the most highly anesthetic steroids. The norsesquiterpenes gyrinidal and gyrinidione were similarly toxic, but possessed little anesthetic action. Steroid activity was highly related to the degree of oxygenation and those oxygenated only at the termini of the molecule were the most active. Miller and Mumma[242] also proposed that water beetle defensive steroids and norsesquiterpenes acted on fish via membrane stabilization and lysis. Both types of compounds exerted their effects on fathead minnows at similar internal concentrations, causing anesthesia at 10^{-6} to 10^{-5}M and death at 10^{-4} to 10^{-3}M. Uptake was a nonmediated process, with the gills absorbing 80% and the skin 20% of steroids removed from solution. Although fish were probably not poisoned by gill-absorbed beetle toxins in a natural setting, the beetle defensive agents were known to be highly repellent to fish, and repellency may have been due to small quantities of gill-absorbed toxins. Zitko *et al.*[243] examined structure-lethality relationships of pyrethrins and pyrethroids and found that lethality to juvenile Atlantic salmon (*Salmo salar*) increased with increasing octanol/water partition coefficient. Lethal thresholds, or geometric mean of lowest concentration with and highest concentration without mortality, were 32.0, 16.5, and 8.8 μg/l for pyrethrin, allethrin, and permethrin, respectively. Actellic, an organophosphoric biocide, was shown by Huda and Svobodova[244] to be highly toxic to fish. The 48-hour LC50 values were 0.005 ml/l for carp, 0.001 ml/l for rainbow trout, and 0.004 ml/l for *Poecilia reticulata*. Minimum lethal concentrations (48-hour LC5) were estimated as 0.001 ml/l actellic for carp, 0.0005 ml/l for rainbow trout, and 0.002 ml/l for *Poecilia reticulata*.

The distribution, metabolism, and effects of the DDT group of insecticides, PCBs, and the cyclodiene insecticides (aldrin, dieldrin, endrin), in aquatic organisms were discussed by Addison.[245] The summarized experimental and field studies suggested that the distribution and retention of these organochlorine compounds (OCs) in environmental samples was controlled by some factors closely related to those governing lipid-water distribution of the OC whether the OC body burden was due to uptake directly from the water or due to uptake by ingestion. No adverse effects were observed by Snarski and Puglisi[246] on survival or growth of first-generation brook trout (*Salvelinus fontinalis*) during a 71-week exposure or on their progeny exposed for 90 days to PCB (Aroclor 1254) concentrations of 0.01 to 0.94 μg/l. However, the PCB tissue concentration was directly proportional to the water concentration, with concentration factors in whole bodies of the trout ranging from 10 000 to 42 000x the water concentration. Steady-state PCB residue levels observed after 14 weeks of exposure were 2 μg/g in fillets of fish at the highest water concentration, and ranged from 0.5 to 9 μg/g in whole bodies of fish exposed to the water concentrations of 0.01 to 0.94 μg/l. Mayer *et al.*[247] investigated the toxicity, bioaccumulation, and biological effects of PCBs in several freshwater invertebrates and fishes. The 96-hour LC50 values for cutthroat trout (*Salmo clarki*) exposed to the Aroclor series 1221–1260 ranged from 1.2 to 61 μg/l. In 30-day continuous-flow tests of Aroclors 1242–1260, rainbow trout was the

TABLE I. A summary of the acute and chronic toxicity of inorganic and organic pollutants to freshwater fish.

Pollutant	Species	Results		Exposure Type[a]	Temp. (°C)	Test Conditions[b]	
Acetanilde[207]	Bluegill (*Lepomis macrochirus*)	96-hr LC50	100 mg/l	S	23	HD pH	55 7.6–7.9
Acetone cyanohydrin[207]	Bluegill	96-hr LC50	0.57 mg/l	S	23	HD pH	55 7.6–7.9
Acetellic[244]	Carp (*Cyprinus carpio*)	48-hr LC50	0.005 ml/l	—	—		—
	Rainbow trout (*Salmo gairdneri*)	48-hr LC50	0.001 ml/l	—	—		—
	Guppy (*Poecilia reticulata*)	48-hr LC50	0.004 ml/l	—	—		—
Aldrin[110]	Striped bass (*Morone saxatilis*)	96-hr LC50	0.010 mg/l	S	20	DO HD	6.0 mg/l 50 mg/l
	Banded killifish (*Fundulus diaphanus*)	96-hr LC50	0.021 mg/l	S	20	pH	7.2
	Pumpkinseed (*Lepomis gibbosus*)	96-hr LC50	0.02 mg/l	S	20		
	White perch (*Roccus americanus*)	96-hr LC50	0.042 mg/l	S	20		
	American eel (*Anguilla rostrata*)	96-hr LC50	0.016 mg/l	S	20		
	Carp	96-hr LC50	0.004 mg/l	S	20		
	Guppy	96-hr LC50	0.02 mg/l	S	20		
Allethrin[243]	Atlantic salmon (*Salmo salar*)	48-hr Lethal threshold concentration (LTC)	16.5 µg/l	S	10		—
Ammonium ferricyanide[207]	Bluegill	96-hr LC50	300 mg/l	S	23	HD pH	55 7.6–7.9
Ammonium picrate[207]	Bluegill	96-hr LC50	220 mg/l	S	23	HD pH	55 7.6–7.9
Amyl alcohol[207]	Bluegill	96-hr LC50	650 mg/l	S	23	HD pH	55 7.6–7.9
Arsenic (sodium arsenate)[132]	Rainbow trout	96-hr LC50	10.8 mg/l	FT	—	ALK DO pH	82–132 4.8–9.0 6.4–8.3
Bayer 73[122]	*Tilapia leucosticta*	3-day-old eggs LC50	8.4–11.4 mg/l	S	25		—
		9-day-old larvae LC50	1.5 mg/l	S	25		—
Bayer 73[123]	Sea lamprey (*Petromyzon marinus*)	24-hr LC50	0.049 mg/l	S	11–18	HD pH	100–136 8.2
	Rainbow trout	24-hr LC50	0.14 mg/l	S	11–18		
Bensulide[115]	Channel catfish (*Ictalurus punctatus*)	96-hr LC50	379 µg/l	S	20–21	ALK HD	80 22
Benzyl alcohol[207]	Bluegill	96-hr LC50	10 mg/l	S	23	HD pH	55 7.6–7.9

TABLE I—(*Continued*)

Pollutant	Species	Results		Exposure Type[a]	Temp. (°C)	Test Conditions[b]	
Bleached kraft mill effluent[220]	Coho salmon (*Oncorhynchus kisutch*)	96-hr LC50	14.2–20.6% waste	S	10–25	DO	Saturation
						pH	6.5
	Rainbow trout	96-hr LC50	14.2–20.6% waste				
Bleached kraft mill effluent[207]	Rainbow trout	96-hr LC50	14–49% waste	S and FT	—	pH	7.5–8.0
Boron (borax)[147]	Channel catfish	Fertilization to 4 days post hatch LC50	155 mg/l	FT	29.4	DO	6.4
						HD	46.7
						pH	8.5
		Fertilization to 4 days post hatch LC50	71 mg/l	FT	29.4	DO	6.5
						HD	194.8
						pH	8.5
	Goldfish (*Carassius auratus*)	Fertilization to 4 days post hatch LC50	65 mg/l	FT	27.0	DO	7.5
						HD	46.2
						pH	8.3
		Fertilization to 4 days post hatch LC50	59 mg/l	FT	27.0	DO	7.5
						HD	194.8
						pH	8.1
	Rainbow trout	Fertilization to 4 days post hatch LC50	27 mg/l	FT	14.0	DO	10.1
						HD	49.0
						pH	7.9
		Fertilization to 4 days post hatch LC50	54 mg/l	FT	13.0	DO	10.3
						HD	191.0
						pH	7.8
Boron (boric acid)[147]	Channel catfish	Fertilization to 4 days post hatch LC50	155 mg/l	FT	25.0	DO	7.3
						HD	51.8
						pH	7.5
		Fertilization to 4 days post hatch LC50	22 mg/l	FT	24.7	DO	7.6
						HD	212.0
						pH	7.6
	Goldfish	Fertilization to 4 days post hatch LC50	46 mg/l	FT	24.8	DO	7.4
						HD	54.5
						pH	7.9
		Fertilization to 4 days post hatch LC50	75 mg/l	FT	24.8	DO	7.5
						HD	207.5
						pH	7.6
	Rainbow trout	Fertilization to 4 days post hatch LC50	100 mg/l	FT	13.7	DO	9.2
						HD	54.1
						pH	7.7
		Fertilization to 4 days post hatch LC50	79 mg/l	FT	13.3	DO	9.6
						HD	204.0
						pH	7.9
Boron (sodium metaborate)[148]	Coho salmon	283-hr LC50	113 mg/l	R	11.0	HD	47
Butyl acetate[207]	Bluegill	96-hr LC50	100 mg/l	S	23	HD	55
						pH	7.6–7.9
Butylamine[207]	Bluegill	96-hr LC50	32 mg/l	S	23	HD	55
						pH	7.6–7.9
Cadmium (cadmium chloride)[169]	Brook trout (*Salvelinus fontinalis*)	60-day MATC†	1–3 μg/l	FT	10	A	3.5
						ALK	30.0
						DO	10.0
						HD	37.0
						pH	6.5–7.2
		60 day MATC	7–12 μg/l	FT	10	A	8.1
						ALK	177.0
						DO	10.6
						HD	188.0
						pH	6.7–7.1

TABLE I—(*Continued*)

Pollutant	Species	Results		Exposure Type[a]	Temp. (°C)	Test Conditions[b]	
	Channel catfish	60-day MATC	11–17 µg/l	FT	22	A	3.0
						ALK	34.0
						DO	7.6
						HD	37.0
						pH	7.5–7.6
		60-day MATC	12–17 µg/l	FT	22	A	4.8
						ALK	172.0
						DO	8.6
						HD	185.0
						pH	7.7–7.8
Cadmium (cadmium chloride)[169] (con't.)	Walleye (*Stizostedion vitreum*)	30-day MATC	9–25 µg/l	FT	15	A	3.8
						ALK	33.0
						DO	10.5
						HD	35.0
						pH	6.8–7.3
		30-day MATC	>86.7 µg/l	FT	15	A	4.8
						ALK	172.0
						DO	8.8
						HD	187.0
						pH	6.9–7.3
Cadmium (cadmium chloride)[170]	Three-spined stickleback (*Gasterosteus aculeatus*)	96-hr LC50	6.5 mg/l	R	15	DO	90–100% Saturation
						HD	110–120
						pH	7.8–8.1
Cadmium (cadmium chloride)[171]	Three-spined stickleback	96-hr LC50	23 mg/l	R	14.6	ALK	99.7
						DO	9.4
						HD	120.6
						pH	8.09
Cadmium (cadmium nitrate)[152]	Rainbow trout	96-hr LC50	6.6 µg/l	FT	—	ALK	82–132
						DO	4.8–9.0
						pH	6.4–8.3
Carbofuran[84]	Mosquitofish (*Gambusia affinis*)	72-hr LC50	0.52 mg/l	S	—		—
	Green sunfish (*Lepomis cyanellus*)	72-hr LC50	0.16 mg/l	S	—		—
Carbon tetrachloride[207]	Bluegill	96-hr LC50	125 mg/l	S	23	HD	55
						pH	7.6–7.9
Chlordane[99]	Fathead minnow (*Pimephales promelas*)	96-hr LC50	36.9 µg/l	FT	25	HD	152
						pH	7.7
	Brook trout	96-hr LC50	47.0 µg/l	FT	15	HD	149
		Lowest effect level	0.32 µg/l	FT	15	pH	7.7
	Bluegill	96-hr LC50	59.0 µg/l	FT	25	HD	161
		MATC	0.54 µg/l	FT	25	pH	7.6
Chlorine (chloramines)[150]	Coho salmon	96-hr LC50	60 µg/l	FT	10–15	ALK	135
						pH	7.5
Chlorine[208]	Bluegill	96-hr LC50	0.44 mg/l	FT	15–32	Intermittent exposure	
Chlorine[270]	Bluegill	96-hr LC50	0.43–0.47 mg/l	FT	6–32	Intermittent exposure	
		Median lethal time (LT50) (exposure 0.52 mg/l) was 19.9 to 74 hrs		FT	6–32	ALK	42–45
						HD	41.5–46.3
						pH	7.25–7.55
Chlorine[142]	Coho salmon	96-hr LC50	0.289 mg/l	S	12	HD	44
	Rainbow trout	96-hr LC50	0.172 mg/l	S	12	pH	7.5

TABLE I—(*Continued*)

Pollutant	Species	Results		Exposure Type[a]	Temp. (°C)	Test Conditions[b]
	Lake trout (*Salvelinus namaycush*)	96-hr LC50	0.200 mg/l	S	12	
	Goldfish	96-hr LC50	1.18 mg/l	S	12	
	Carp	96-hr LC50	0.800 mg/l	S	12	
	Fathead minnow	96-hr LC50	0.998 mg/l	S	12	
	White sucker (*Catostomus commersoni*)	96-hr LC50	0.379 mg/l	S	12	
	Black bullhead (*Ictalurus melas*)	96-hr LC50	1.41 mg/l	S	12	
	Channel catfish	96-hr LC50	0.156 mg/l	S	12	
	Green sunfish	96-hr LC50	1.28 mg/l	S	12	
	Bluegill	96-hr LC50	0.555 mg/l	S	12	
	Yellow perch (*Perca flavescens*)	96-hr LC50	0.558 mg/l	S	12	
Chloropyrifos[94]	Green sunfish	72-hr LC50	0.04 mg/l	S	—	—
	Mosquitofish	72-hr LC50	0.26 mg/l	S	—	—
Chromium[223]	Rainbow trout	96-hr LC50	11.2 mg/l	—	—	—
		96-hr LC50 (for trout exposed previously to low level PCB = 0.01 µg/l for 30 days)	9.0 mg/l	—	—	—
		96-hr LC50 (for trout exposed previously to high level PBC = 0.1 µg/l for 30 days)	7.05 mg/l	—	—	—
Chromium (chromium nitrate)[102]	Rainbow trout	96-hr LC50	24.09 mg/l	FT	—	ALK 82–132 DO 4.8–9.0 pH 6.4–8.3
Chromium (sodium dichromate)[100]	Rainbow trout	60-day MATC	51–105 µg/l	FT	10	A 3.3 ALK 30.1 DO 9.1 HD 33.4 pH 6.7–7.0
	Lake trout	60-day MATC	105–194 µg/l	FT	10	A 3.6 ALK 31.5 DO 9.5 HD 34.0 pH 6.8–7.1
	Channel catfish	60-day MATC	150–305 µg/l	FT	22	A 4.0 ALK 33.7 DO 8.1 HD 36.2 pH 7.0–7.4
Chromium (sodium dichromate)[102] (con't.)	Bluegill	60-day MATC	522–1 122 µg/l	FT	25	A 6.6 ALK 33.0 DO 6.6 HD 38.3 pH 6.7–7.1
	White sucker	60-day MATC	290–538 µg/l	FT	17	A 3.2 ALK 34.6 DO 8.9 HD 38.8 pH 6.9–7.2

TABLE I—(*Continued*)

Pollutant	Species	Results		Exposure Type[a]	Temp. (°C)	Test Conditions[b]
	Northern pike (*Esox lucius*)	20-day MATC	538–963 µg/l	FT	17	A 3.3 ALK 35.6 DO 9.0 HD 37.8 pH 6.7–7.0
	Walleye	30-day MATC	>2 161 µg/l	FT	15	A 3.4 ALK 33.8 DO 9.5 HD 38.5 pH 6.8–7.2
Copper (copper sulfate)[184]	Rainbow trout	240-hr LC20	22 µg/l	—	12	HD 30 pH 6
		240-hr LC20	300 µg/l	—	12	HD 360 pH 8
Copper (copper sulfate)[180]	Brook trout	60-day MATC	3–5 µg/l	FT	10	A 3.3 ALK 27.8 DO 10.0 HD 37.5 pH 6.6–7.1
Copper (copper sulfate)[180] (con't.)	Brook trout	60-day MATC	5–8 µg/l	FT	10	A 8.3 ALK 177.6 DO 11.0 HD 187.0 pH 6.7–7.1
	Channel catfish	60-day MATC	12–18 µg/l	FT	22	A 3.2 ALK 34.1 DO 7.3 HD 36.0 pH 7.4–7.6
		60-day MATC	13–19 µg/l	FT	22	A 4.8 ALK 172.9 DO 8.7 HD 186.3 pH 7.5–7.8
	Walleye	30-day MATC	13–21 µg/l	FT	15	A 3.3 ALK 34.0 DO 9.9 HD 35.0 pH 6.8–7.3
		30-day MATC	>71 µg/l	FT	15	A 4.6 ALK 176.0 DO 8.9 HD 189.0 pH 7.0–7.3
Copper (copper sulfate)[240]	Rainbow trout	96-hr LC50	0.102 mg/l	FT	15	ALK 200–255
	Zebrafish (*Brachydanio rerio*)	96-hr LC50	0.149 mg/l	FT	25	HD 350–375 pH 8.0–8.3
	Flagfish (*Jordanella floridae*)	96-hr LC50	1.27 mg/l	FT	25	
Copper (cupric chloride)[130]	Coho salmon	96-hr LC50	Nov.—74 µg/l Mar.—70 µg/l May—60 µg/l (smoltification)	R R R	10–12 10–12 10–12	ALK 68–78 DO 8.1–10.7 HD 89–99 pH 6.74–7.96
Copper (cupric nitrate)[182]	Rainbow trout	96-hr LC50	0.253 mg/l	FT	—	ALK 82–132 DO 4.8–9.0 pH 6.4–8.3

TABLE I—(*Continued*)

Pollutant	Species	Results		Exposure Type[a]	Temp. (°C)	Test Conditions[b]	
Crotonaldehyde[207]	Bluegill	96-hr LC50	3.5 mg/l	S	23	HD pH	55 7.6–7.9
Cyanide[522]	Rainbow trout	96-hr LC50	90.0 µg/l	—	—		—
		96-hr LC50 (for trout exposed previously to low levels of PCB—0.01 µg/l for 30 days)	66.2 µg/l	—	—		—
		96-hr LC50 (for trout exposed previously to high levels of PCB—0.1 µg/l for 30 days)	65.8 µg/l	—	—		—
Cyanide (hydrogen cyanide)[162]	Fathead minnow	MATC	12.9–19.6 µg/l	FT	25	DO pH	5.9–6.3 8.06–8.09
Cyanogen bromide[207]	Bluegill	96-hr LC50	0.24 mg/l	S	23	HD pH	55 7.6–7.9
Cyclohexanol[207]	Bluegill	96-hr LC50	1 100 mg/l	S	23	HD pH	55 7.6–7.9
2,4-D[116]	Striped bass	96-hr LC50	70.1 mg/l	S	20	DO	6.0
	Banded killifish	96-hr LC50	26.7 mg/l	S	20	HD	50
	Pumpkinseed	96-hr LC50	94.6 mg/l	S	20	pH	7.2
	White perch	96-hr LC50	40.0 mg/l	S	20		
	American eel	96-hr LC50	300.6 mg/l	S	20		
	Carp	96-hr LC50	96.5 mg/l	S	20		
	Guppy	96-hr LC50	70.7 mg/l	S	20		
DDT[92,93]	Fathead minnow	DDT in food MATC	0.9 µg/l	FT	25	A ALK HD	2.8 42.5 43.9
		DDT in food and water MATC	0.4 µg/l	FT	25	pH	7.2–7.8
Diacetone alcohol[207]	Bluegill	96-hr LC50	420 mg/l	S	23	HD pH	55 7.6–7.9
Dialkyl tetralin-indane sulfonate C_{18}[227]	Fathead minnow	24-hr LC50	87.0 mg/l	S	—	HD	100
		48-hr LC50	86.1 mg/l	S	—		
Dialkyl tetralin-indane[227] sulfonate C_{12}[227]	Fathead minnow	24-hr LC50	24.8 mg/l	S	—	HD	100
		48-hr LC50	21.5 mg/l	S	—		
Dialkyl tetralin-indane sulfonate C_{14}[227]	Fathead minnow	24-hr LC50	8.1 mg/l	S	—	HD	100
		48-hr LC50	5.3 mg/l	S	—		
o-Dichlorobenzene[207]	Bluegill	96-hr LC50	27 mg/l	S	23	HD pH	55 7.6–7.9
Dichloroethane[207]	Bluegill	96-hr LC50	550 mg/l	S	23	HD pH	55 7.6–7.9
Dichloropropane[207]	Bluegill	96-hr LC50	320 mg/l	S	23	HD pH	55 7.6–7.9
Diethylene glycol monobutyl ether[207]	Bluegill	96-hr LC50	1 300 mg/l	S	23	HD pH	55 7.6–7.9
Diethylene glycol monoethyl ether[207]	Bluegill	96-hr LC50	10 000 mg/l	S	23	HD pH	55 7.6–7.9
Dimethyl sulfate[207]	Bluegill	96-hr LC50	7.5 mg/l	S	23	HD pH	55 7.6–7.9
Dioxane[207]	Bluegill	96-hr LC50	10 000 mg/l	S	23	HD pH	55 7.6–7.9

TABLE I—(*Continued*)

Pollutant	Species	Results		Exposure Type[a]	Temp. (°C)	Test Conditions[b]	
Distillery waste[208]	*Puntius sophore*	96-hr LC50	8.1% waste	S	20–24	ALK	160
		96-hr LC50	6.3% waste	S	30–34	DO	7.2
						HD	110
						pH	6.2–6.5
	Mystus vittatus	96-hr LC50	11.15% waste	S	20–24	ALK	160
		96-hr LC50	10.0% waste	S	30–34	DO	7.2
						HD	110
						pH	6.1–6.4
Diterpene[204]	Sockeye salmon (*Oncorhynchus nerka*)	96-hr LC50	2.2 mg/l	S	4–7	DO	Near saturation
						pH	7–7.3
DNBP[115]	Channel catfish	96-hr LC50	118 μg/l	S	20–21	ALK	80
						HD	22
Dodecyl sodium sulfate[240]	Rainbow trout	96-hr LC50	4.62 mg/l	FT	15	ALK	200–225
	Zebrafish	96-hr LC50	7.97 mg/l	FT	25	HD	350–375
	Flagfish	96-hr LC50	8.10 mg/l	FT	25	pH	8.0–8.3
Epichlorohydrin[207]	Bluegill	96-hr LC50	35 mg/l	S	23	HD	55
						pH	7.6–7.9
Ethyl ether[207]	Bluegill	96-hr LC50	10 000 mg/l	S	23	HD	55
						pH	7.6–7.9
Ethylene glycol diacetate[207]	Bluegill	96-hr LC50	90 mg/l	S	23	HD	55
						pH	7.6–7.9
Ethylene glycol monobutyl[207] ether[207]	Bluegill	96-hr LC50	1 460 mg/l	S	23	HD	55
						pH	7.6–7.9
Ethylene glycol monoethyl ether[207]	Bluegill	96-hr LC50	10 000 mg/l	S	23	HD	55
						pH	7.6–7.9
Ethylene glycol monoethyl ether acetate[207]	Bluegill	96-hr LC50	45 mg/l	S	23	HD	55
						pH	7.6–7.9
Ethylene glycol monomethyl ether[207]	Bluegill	96-hr LC50	10 000 mg/l	S	23	HD	55
						pH	7.6–7.9
Forest fire retardant:[50] Fire-Trol 100	Coho salmon	96-hr LC50	90 to >1 500 mg/l	S	6–11	pH	6.9–7.1
		96-hr LC50	280 mg/l	FT	6–11		
		20-da LC50	73 mg/l	FT	6–11		
	Rainbow trout	96-hr LC50	150 to >1 000 mg/l	S	6–11		
		96-hr LC50	>100 mg/l	FT	6–11		
		20-da LC50	43 mg/l	FT	6–11		
	Bluegill	96-hr LC50	>1 500 mg/l	S	21		
	Fathead minnow	96-hr LC50	>1 500 mg/l	S	21		
	Largemouth bass	96-hr LC50	>1 500 mg/l	S	21		
Fire-Trol 931	Coho salmon	96-hr LC50	580–1 000 mg/l	S	6–11	pH	6.6–7.0
	Rainbow trout	96-hr LC50	700–1 000 mg/l	S	6–11		
	Bluegill	96-hr LC50	>1 500 mg/l	S	21		
	Fathead minnow	96-hr LC50	>1 500 mg/l	S	21		
	Largemouth bass	96-hr LC50	>1 500 mg/l	S	21		
Phos-Chek 202	Coho salmon	96-hr LC50	145–300 mg/l	S	6–11	pH	7.3–7.7
	Rainbow trout	96-hr LC50	105–230 mg/l	S	6–11		
	Bluegill	96-hr LC50	840 mg/l	S	21		
	Fathead minnow	96-hr LC50	650 mg/l	S	21		
	Largemouth bass	96-hr LC50	840 mg/l	S	21		
Phos-Chek 259	Coho salmon	96-hr LC50	145–250 mg/l	S	6–11	pH	7.4–7.7
	Rainbow trout	96-hr LC50	94–165 mg/l	S	6–11		
	Bluegill	96-hr LC50	350 mg/l	S	21		

TABLE I—(*Continued*)

Pollutant	Species	Results		Exposure Type[a]	Temp. (°C)	Test Conditions[b]	
	Fathead minnow	96-hr LC50	300 mg/l	S	21		
	Largemouth bass	96-hr LC50	450 mg/l	S	21		
Formalin[129]	Rainbow trout	96-hr LC50	118 µl/l	FT	12	HD	20
	Atlantic salmon	96-hr LC50	173 µl/l	FT	12	pH	6.5
	Lake trout	96-hr LC50	100 µl/l	FT	12		
	Black bullhead	96-hr LC50	62.1 µl/l	FT	12		
	Channel catfish	96-hr LC50	65.8 µl/l	FT	12		
	Green sunfish	96-hr LC50	173 µl/l	FT	12		
	Bluegill	96-hr LC50	100 µl/l	FT	12		
	Smallmouth bass (*Micropterus dolomieui*)	96-hr LC50	136 µl/l	FT	12		
	Largemouth bass (*Micropterus salmoides*)	96-hr LC50	143 µl/l	FT	12		
Furanace[138]	Atlantic salmon	96-hr LC50	1.41 mg/l	S	12	HD	44
	Rainbow trout	96-hr LC50	1.00 mg/l	S	12		
	Fathead minnow	96-hr LC50	0.820 mg/l	S	12		
	Channel catfish	96-hr LC50	1.07 mg/l	S	12		
	Green sunfish	96-hr LC50	2.48 mg/l	S	12		
	Bluegill	96-hr LC50	3.00 mg/l	S	12		
Heptachloronorbornene[65]	Fathead minnow	96-hr LC50	85.6 µg/l	FT	25	A	1.5–2.5
						ALK	42–43
						DO	7.2–8.6
						HD	45–47
						pH	7.2–7.7
Hexachlorocyclopentadiene[65]	Fathead minnow	96-hr LC50	7.0 µg/l	FT	25		
Hexachloronorbornadiene[65]	Fathead minnow	96-hr LC50	188 µg/l	FT	25		
Hexylene glycol[207]	Bluegill	96-hr LC50	10 000 mg/l	S	23	HD	55
						pH	7.6–7.9
Hydrogen sulfide[50]	Fathead minnow	96-hr LC50	0.0107–0.7754 mg/l	FT	6–25	HD	220
						pH	7.9
		MATC	3.6–10.3 µg/l	FT	22–25	DO	6.1–7.4
						HD	220
						pH	7.7–7.9
Imidan[90]	Chinook salmon (*Oncorhynchus tshawytscha*)	96-hr LC50	150 µg/l	S	10	ALK	35
						HD	40
						pH	7.2
	Bluegill	96-hr LC50	70 µg/l	S	20		
	Smallmouth bass	96-hr LC50	150 µg/l	S	20		
	Rainbow trout						
	Fingerling	96-hr LC50	560 µg/l	S	10		
	Yolk sac fry	96-hr LC50	>10 000 µg/l	S	10		
	Eyed eggs	96-hr LC50	>10 000 µg/l	S	10		
	Fathead minnow	96-hr LC50	7 300 µg/l	S	20		
	Channel catfish	96-hr LC50	11 000 µg/l	S	20		
Iron (lime-neutralized hydroxide suspensions)[184]	Coho salmon	90-day MATC	0.97–1.27 mg/l	FT	11	DO	97–100% Saturation
						HD	162–180
						pH	8.03–8.12
Isodecyl diphenyl phosphate[207]	Bluegill	96-hr LC50	6 700 mg/l	S	23	HD	55
						pH	7.6–7.9

TABLE I—(*Continued*)

Pollutant	Species	Results		Exposure Type[a]	Temp. (°C)	Test Conditions[b]
LAS:[226]						
0% biodegraded	Bluegill	96-hr LC50	0.72 mg/l	S	20.8	ALK 40.0 DO 5.8 HD 54.0 pH 7.2
36.7% biodegraded	Bluegill	96-hr LC50	0.89 mg/l	S	20.5	ALK 39.5 DO 6.2 HD 62.0 pH 7.4
53.3% biodegraded	Bluegill	96-hr LC50	1.16 mg/l	S	20.6	ALK 40.0 DO 6.2 HD 49.6 pH 7.3
76.0% biodegraded	Bluegill	96-hr LC50	1.64 mg/l	S	21.0	ALK 58.0 DO 4.9 HD 61.0 pH 7.4
LAS:[227]						
Commercial	Fathead minnow	24-hr LC50	1.9 mg/l	S	—	HD 100
		48-hr LC50	1.7 mg/l	S	—	
Homolog C_{10}	Fathead minnow	24-hr LC50	48.0 mg/l	S	—	HD 100
		48-hr LC50	43.0 mg/l	S	—	
Homolog C_{11}	Fathead minnow	24-hr LC50	17.0 mg/l	S	—	HD 100
		48-hr LC50	16.0 mg/l	S	—	
Homolog C_{12}	Fathead minnow	24-hr LC50	4.7 mg/l	S	—	HD 100
		48-hr LC50	4.7 mg/l	S	—	
Homolog C_{13}	Fathead minnow	24-hr LC50	1.7 mg/l	S	—	HD 100
		48-hr LC50	0.4 mg/l	S	—	
Homolog C_{14}	Fathead minnow	24-hr LC50	0.6 mg/l	S	—	HD 100
		48-hr LC50	0.4 mg/l	S	—	
Model intermediate C_4	Fathead minnow	24-hr LC50	10 000 mg/l	S	—	HD 100
		48-hr LC50	10 000 mg/l	S	—	
Model intermediate C_5	Fathead minnow	24-hr LC50	10 000 mg/l	S	—	HD 100
		48-hr LC50	10 000 mg/l	S	—	
Model intermediate C_{11}	Fathead minnow	24-hr LC50	85.9 mg/l	S	—	HD 100
		48-hr LC50	76.6 mg/l	S	—	
Lead (lead nitrate)[182]	Rainbow trout	96-hr LC50	8.0 mg/l	FT	—	ALK 82–132 DO 4.8–9.0 pH 6.4–8.3
Lead (lead nitrate)[180]	Rainbow trout	60-day MATC	71–146 µg/l	FT	10	A 3.5 ALK 30.2 DO 10.3 HD 34.6 pH 6.9–7.4
	Lake trout	60-day MATC	48–83 µg/l	FT	10	A 3.4 ALK 29.6 DO 9.7 HD 32.6 pH 7.0–7.3
	Channel catfish	60-day MATC	75–136 µg/l	FT	22	A 3.4 ALK 33.6 DO 8.5 HD 36.0 pH 6.8–7.3
	Bluegill	60-day MATC	70–120 µg/l	FT	25	A 5.3 ALK 33.3 DO 6.9 HD 40.7 pH 6.7–7.2

TABLE I—(*Continued*)

Pollutant	Species	Results		Exposure Type[a]	Temp. (°C)	Test Conditions[b]	
	White sucker	60-day MATC	119–253 µg/l	FT	17	A	3.5
						ALK	34.8
						DO	8.8
						HD	37.6
						pH	6.7–7.1
Lead (lead nitrate)[100] (con't.)	Northern pike	20-day MATC	253–483 µg/l	FT	17	A	3.6
						ALK	33.1
						DO	8.7
						HD	35.4
						pH	6.7–7.3
	Walleye	30-day MATC	237–397 µg/l	FT	15	A	3.7
						ALK	33.8
						DO	10.3
						HD	37.6
						pH	6.7–7.1
Malachite green[127]	Chinook salmon	96-hr LC50	0.224 mg/l	FT	12	HD	20
	Coho salmon	96-hr LC50	0.383 mg/l	FT	12	pH	7.5
	Atlantic salmon	96-hr LC50	0.283 mg/l	FT	12		
	Brown trout (*Salmo trutta*)	96-hr LC50	0.237 mg/l	FT	12		
	Rainbow trout	96-hr LC50	0.248 mg/l	FT	12		
	Brook trout	96-hr LC50	0.220 mg/l	FT	12		
	Channel catfish	96-hr LC50	0.112 mg/l	FT	12		
	Largemouth bass	96-hr LC50	0.0728 mg/l	FT	12		
	Smallmouth bass	96-hr LC50	0.0453 mg/l	FT	12		
	Bluegill	96-hr LC50	0.0305 mg/l	FT	12		
Malathion[118]	Striped bass	96-hr LC50	0.039 mg/l	S	20	DO	6.0
	Banded killifish	96-hr LC50	0.24 mg/l	S	20	HD	50
	Pumpkinseed	96-hr LC50	0.48 mg/l	S	20	pH	7.2
	White perch	96-hr LC50	1.1 mg/l	S	20		
	American eel	96-hr LC50	0.50 mg/l	S	20		
	Carp	96-hr LC50	1.9 mg/l	S	20		
	Guppy	96-hr LC50	1.2 mg/l	S	20		
Malinate[84]	Mosquitofish	72-hr LC50	17.0 mg/l	S	—		—
	Green sunfish	72-hr LC50	34.5 mg/l	S	—		—
Mercury (mercurous nitrate)[132]	Rainbow trout	96-hr LC50	33.0 µg/l	FT	—	ALK	82–132
						DO	4.8–9.0
						pH	6.4–8.3
Methanesulfonyl chloride[207]	Bluegill	96-hr LC50	11 mg/l	S	23	HD	55
						pH	7.6–7.9
Methoxychlor[26]	Atlantic salmon	24-hr LC50	75–92 µg/l	S	2–20	DO	8.2–14.6
						pH	6.7–7.3
Methyl bromide[207]	Bluegill	96-hr LC50	11 mg/l	S	23	HD	55
						pH	7.6–7.9
Methyl chloride[207]	Bluegill	96-hr LC50	550 mg/l	S	23	HD	55
						pH	7.6–7.9
Methyl parathion[118]	Striped bass	96-hr LC50	14.0 mg/l	S	20	DO	6.0
	Banded killifish	96-hr LC50	15.2 mg/l	S	20	HD	50
	Pumpkinseed	96-hr LC50	3.6 mg/l	S	20	pH	7.2
	White perch	96-hr LC50	14.0 mg/l	S	20		
	American eel	96-hr LC50	6.3 mg/l	S	20		
	Carp	96-hr LC50	14.8 mg/l	S	20		
	Guppy	96-hr LC50	6.2 mg/l	S	20		
Morpholine[207]	Bluegill	96-hr LC50	350 mg/l	S	23	HD	55
						pH	7.6–7.9
Naled[26]	Atlantic salmon	24-hr LC50	165 µg/l	S	16–20	DO	4.4–7.2
Nickel (nickel nitrate)[132]	Rainbow trout	96-hr LC50	35.5 mg/l	FT	—	ALK	82–132
						DO	4.8–9.0
						pH	6.4–8.3

TABLE I—(*Continued*)

Pollutant	Species	Results		Exposure Type[a]	Temp. (°C)	Test Conditions[b]	
Nitrite (sodium nitrite)[153]	Chinook salmon	48-hr LC50	19 mg/l (freshwater)	S	9.1	pH	7.8
		48-hr, 90% survival (seawater)	1 070 mg/l	S	9.1	pH	7.6
m-Nitrochlorobenzene[307]	Bluegill	96-hr LC50	1.2 mg/l	S	23	HD	55
						pH	7.6–7.9
Noxfish (5% rotenone)[125]	Bowfin (*Amia calva*)	96-hr LC50	30.0 µg/l	S	12	pH	7.5
	Coho salmon	96-hr LC50	62.0 µg/l	S	12		
	Chinook salmon	96-hr LC50	36.9 µg/l	S	12		
	Rainbow trout	96-hr LC50	46.0 µg/l	S	12		
	Atlantic salmon	96-hr LC50	21.5 µg/l	S	12		
	Brook trout	96-hr LC50	44.3 µg/l	S	12		
	Lake trout	96-hr LC50	26.9 µg/l	S	12		
	Northern pike	96-hr LC50	33.0 µg/l	S	12		
	Goldfish	96-hr LC50	497 µg/l	S	12		
	Carp	96-hr LC50	50.0 µg/l	S	12		
	Fathead minnow	96-hr LC50	142 µg/l	S	12		
	Longnose sucker (*Catostomus catostomus*)	96-hr LC50	57.0 µg/l	S	12		
	White sucker	96-hr LC50	68.0 µg/l	S	12		
	Black bullhead	96-hr LC50	389 µg/l	S	12		
	Channel catfish	96-hr LC50	164 µg/l	S	12		
	Green sunfish	96-hr LC50	141 µg/l	S	12		
	Bluegill	96-hr LC50	141 µg/l	S	12		
	Smallmouth bass	96-hr LC50	79.0 µg/l	S	12		
	Largemouth bass	96-hr LC50	142 µg/l	S	12		
	Yellow perch	96-hr LC50	70.0 µg/l	S	12		
	Walleye	24-hr LC50	16.5 µg/l	S	12		
Parathion[84]	Mosquitofish	72-hr LC50	0.20 mg/l	S	—		—
	Green sunfish	72-hr LC50	0.02 mg/l	S	—		—
PCB:[247]							
Arochlor 1 221	Cutthroat trout (*Salmo clarki*)	96-hr LC50	1.2 mg/l	S	9	ALK	35
						pH	7.1
Arochlor 1 232	Cutthroat trout	96-hr LC50	2.5 mg/l	S	9	ALK	35
						pH	7.1
Arochlor 1 242	Rainbow trout	25-day LC50	12 µg/l	FT	17	ALK	237
	Bluegill	30-day LC50	84 µg/l	FT	17	pH	7.4
	Channel catfish	30-day LC50	87 µg/l	FT	17		
	Cutthroat trout	96-hr LC50	5.4 mg/l	S	9	ALK	35
						pH	7.1
Arochlor 1 248	Rainbow trout	25-day LC50	3.4 µg/l	FT	17	ALK	237
	Bluegill	30-day LC50	78 µg/l	FT	17	pH	7.4
	Channel catfish	30-day LC50	75 µg/l	FT	17		
	Cutthroat trout	96-hr LC50	5.7 mg/l	S	9	ALK	35
						pH	7.1
Arochlor 1 254	Rainbow trout	25-day LC50	27 µg/l	FT	17	ALK	237
	Bluegill	30-day LC50	177 µg/l	FT	17	pH	7.4
	Channel catfish	30-day LC50	139 µg/l	FT	17		
	Cutthroat trout	96-hr LC50	42 mg/l	S	9	ALK	35
						pH	7.1
Arochlor 1 260	Rainbow trout	30-day LC50	51 µg/l	FT	17	ALK	237
	Bluegill	30-day LC50	400 µg/l	FT	17	pH	7.4
	Channel catfish	30-day LC50	433 µg/l	FT	17		
	Cutthroat trout	96-hr LC50	61 mg/l	S	9	ALK	35
						pH	7.1

TABLE I—(*Continued*)

Pollutant	Species	Results		Exposure Type[a]	Temp. (°C)	Test Conditions[b]	
Pentachlorphenate (sodium pentachlorophenate)[240]	Rainbow trout	96-hr LC50	0.23 mg/l	FT	15	ALK	200–225
	Zebrafish	96-hr LC50	1.08 mg/l	FT	25	HD	350–375
	Flagfish	96-hr LC50	1.74 mg/l	FT	25	pH	8.0–8.3
Pentachlorophenol[257]	Bluegill	96-hr LC50	0.3 mg/l	S	17–21	DO	7.4–8.6
						pH	7.2–7.7
Permethrin[242]	Atlantic salmon	48-hr LTC	8.8 µg/l	S	10		—
Phenol[240]	Rainbow trout	96-hr LC50	11.6 mg/l	FT	15	ALK	200–225
	Zebrafish	96-hr LC50	29.0 mg/l	FT	25	HD	350–375
	Flagfish	96-hr LC50	36.3 mg/l	FT	25	pH	8.0–8.3
Picloram[240]	Rainbow trout	96-hr LC50	26.0 mg/l	FT	15	ALK	200–225
	Zebrafish	96-hr LC50	35.5 mg/l	FT	25	HD	350–375
	Flagfish	96-hr LC50	26.1 mg/l	FT	25	pH	8.0–8.3
Polypropylene glycol[207]	Bluegill	96-hr LC50	1 700 mg/l	S	23	HD	55
						pH	7.6–7.9
Propanil[24]	Mosquitofish	72-hr LC50	7.62 mg/l	S	—		—
	Green sunfish	72-hr LC50	5.85 mg/l	S	—		—
Propanil[115]	Channel catfish	96-hr LC50	3 796 µg/l	S	20–21	ALK	80
						HD	22
Propionaldehyde[207]	Bluegill	96-hr LC50	130 mg/l	S	23	HD	55
						pH	7.6–7.9
Pulp mill foam[266]	Sockeye salmon	96-hr LC50	0.01–0.017% waste	S	4–7	DO	Near saturation
						pH	7.0–7.3
Pyrethrins[242]	Atlantic salmon	48-hr LTC	32.0 µg/l	S	10		—
Resin acid[266]	Sockeye salmon	96-hr LC50	1.3–3.9 mg/l	S	4–7	DO	Near saturation
						pH	7.0–7.3
Sewage (chlorinated municipal wastes)[278]	Rainbow trout	96-hr LC50	<15.3–70.9% waste	FT	15		—
		96-hr LC50	<15.3–62.4% waste	S	15		—
	Black bullhead	96-hr LC50	<15.3% waste	S	15		—
Silver (silver nitrate)[152]	Rainbow trout	96-hr LC50	28.8 µg/l	FT	—	ALK	82–132
						DO	4.8–9.0
						pH	6.4–8.3
Sodium fluorosilicate[207]	Bluegill	96-hr LC50	65 mg/l	S	23	HD	55
						pH	7.6–7.9
Sodium lauryl sulphate[288]	Brown trout	Median Survival Time (MST) =					
		0.07-hr	1 000 mg/l	R	15	HD	25
		0.07-hr	560 mg/l	R	15		
		0.08-hr	320 mg/l	R	15		
		0.15-hr	180 mg/l	R	15		
		0.26-hr	150 mg/l	R	15		
		0.86-hr	120 mg/l	R	15		
		2.15-hr	100 mg/l	R	15		
		6.5-hr	56 mg/l	R	15		
		32-hr	32 mg/l	R	15		
		45-hr	18 mg/l	R	15		
Strychnine[207]	Bluegill	96-hr LC50	0.87 mg/l	S	23	HD	55
						pH	7.6–7.9
2,4,5-T[116]	Striped bass	96-hr LC50	14.6 mg/l	S	20	DO	6.0
	Banded killifish	96-hr LC50	17.4 mg/l	S	20	HD	50
	Pumpkinseed	96-hr LC50	20.0 mg/l	S	20	pH	7.2
	White perch	96-hr LC50	16.4 mg/l	S	20		
	American eel	96-hr LC50	43.7 mg/l	S	20		
	Carp	96-hr LC50	41.1 mg/l	S	20		
	Guppy	96-hr LC50	28.1 mg/l	S	20		

TABLE I—(*Continued*)

Pollutant	Species	Results		Exposure Type[a]	Temp. (°C)	Test Conditions[b]	
Tetramethyl-lead[207]	Bluegill	96-hr LC50	84 mg/l	S	23	HD	55
						pH	7.6–7.9
TFM[183]	Sea lamprey	24-hr LC50	0.78 mg/l	S	11–18	HD	100–136
	Rainbow trout	24-hr LC50	3.85 mg/l	S	11–18	pH	8.2
Thallium acetate[207]	Bluegill	96-hr LC50	170 mg/l	S	23	HD	55
						pH	7.6–7.9
Tricresylphos-phate[207]	Bluegill	96-hr LC50	7 000 mg/l	S	23	HD	55
						pH	7.6–7.9
Triethylene glycol[207]	Bluegill	96-hr LC50	10 000 mg/l	S	23	HD	55
						pH	7.6–7.9
Trifluralin[113]	Channel catfish	96-hr LC50	417 μg/l	S	20–21	ALK	80
						HD	22
Triphenylphos-phate[207]	Bluegill	96-hr LC50	290 mg/l	S	23	HD	55
						pH	7.6–7.9
Vinylidene chloride[207]	Bluegill	96-hr LC50	220 mg/l	S	23	HD	55
						pH	7.6–7.9
Xylene[113]	Rainbow trout	96-hr LC50	13.5 mg/l	S	12		—
Zinc (zinc acetate)[182]	Rainbow trout	96-hr LC50	0.55 mg/l	FT	—	ALK	82–132
						DO	4.8–9.0
						pH	6.4–8.3
Zinc (zinc chloride)[150]	Coho salmon	96-hr LC50	4.6 mg/l	R	10–12	ALK	68–78
						DO	8.1–10.7
						HD	89–99
						pH	6.74–7.96
Zinc (zinc sulfate)[172]	Sockeye salmon	LT50 for 1 mg/l exposure Infected with cestode = 39.4 hrs Non-infected = 52.8 hrs		FT	10	HD	13
Zinc (zinc sulfate)[150]	Rainbow trout	85 days significant growth inhibition	1.12 mg/l	FT	10	DO	7.4–8.7
						HD	374
						pH	7.3

[a] FT = flow-through bioassay; S = static bioassay; R = renewal bioassay.
[b] MATC = Maximum Acceptable Toxicant Concentration.
[c] A = acidity, mg/l; ALK = alkalinity, mg/l; DO = dissolved oxygen, mg/l; HD = hardness mg/l.

most susceptible species to PCBs, followed by bluegill and channel catfish; LC50s ranged from 3.0 to 422 μg/l. Channel catfish accumulated Aroclors 1248 and 1254 to levels that were 56 370 and 61 190x the levels in the water, respectively, and only Aroclor 1254 altered thyroid activity in channel catfish fed Aroclors 1232, 1248, 1254, or 1260. Dietary exposures of coho salmon (*Oncorhynchus kisutch*) to Aroclor 1254 at rates of 1.45 to 14 500 μg/kg body weight per day had no effect on growth, but all fish treated with the highest concentration died after 260 days. The distribution and elimination of 2,5,2′,5′-[^{14}C] tetrachlorobiphenyl (TCB) in rainbow trout over an extended period of time were examined by Guiney *et al.*[348] Carcass, muscle, skin, lower gastrointestinal tract, and fat of fish exposed to [^{14}C] TCB for 36 hours contained 88% of the ^{14}C. Upon transfer to clean water approximately 30% of the [^{14}C] TCB was eliminated after 2 weeks (half-life of 1.55 days), but only a 6% loss was observed in the following 126 days (half-life of 2.66 years), while the bile and blood, which initially had high concentrations of [^{14}C] TCB, eliminated the compound rapidly and nearly completely. Zitko and Hutzinger[349] examined the accumulation of halogenated hydrocarbons by Atlantic salmon and found no major differences between the accumulation of di- and tetra-, bromo- and chlorobiphenyls. However, in contrast to hexachlorobenzene, hexabromobenzene did not accumulate either from water or from food. The fish accumulated higher concentrations of both chloro- and bromo- substituted compounds from water than from food, and in both groups of compounds the accumulation coef-

ficients (during the uptake from water) generally decreased with increasing degree of substitution, but increased with increasing degree of substitution when taken up from food. Zitko and Carson[250] compared the uptake of Arochlor 1242 and Choralkylene 12 from food and water by juvenile Atlantic salmon. The smaller accumulation coefficients of Chloralkylene 12 (78 for water, 0.230 for food) compared to those of Arochlor 1242 (600 and 0.629, respectively), and shorter excretion half-lives of Chloralkylene 12 (71 hours following uptake from water, 28 days following uptake from food) compared to Arochlor 1242 (207 hours and 35 days, respectively), were contrary to the expected degree of accumulation based on lipid solubility. It was considered likely that isopropylated chlorobiphenyls, such as Chloralkylene 12, were subject to oxidative degradation resulting in the formation of more water-soluble products, and that this contributed to the lower accumulation and faster excretion of Chloralkylene 12 in comparison with Arochlor 1242. Zitko[251] also compared the accumulation of the commercial polybrominated biphenyls (PBBs), BP-6 and OB, with that of the PCB, Aroclor 1254, from food and from water by Atlantic salmon. Arochlor 1254 was accumulated from water much more than the PBBs, but the accumulation of bromobiphenyls from food was higher or equal to that of Arochlor 1254. The accumulation coefficients decreased with increasing number of bromine atoms in the PBBs, and only those brominated biphenyls with six or fewer bromine atoms were accumulated from water.

Eels exposed to crude oil suspensions in water by Ogata *et al.*[252] accumulated paraffins, particularly C_{13} to C_{15} compounds, and aromatic hydrocarbons. Because the eels also accumulated crude oil sulfur compounds, these compounds may be used as a marker of fish oil pollution. Manner and Muehleman[253] measured the uptake of tritiated uridine by five different developmental stages of fathead minnow embryos with and without treatment in a 15 μg/l solution of LAS. The embryos with LAS treatment displayed lower radiation counts at all ages compared to controls, indicating an inhibition of diffusion and uptake of ^{3}H-uridine and/or RNA synthesis, due probably to LAS-induced chemical or morphological changes in the chorionic membrane. In a study conducted by Mayer,[254] fathead minnows continuously exposed to a concentration range of 1.9 to 62 μg/l di-2-ethylhexyl phthalate (DEHP) for 56 days had accumulation factors ranging downward from 886 to 122 as exposure concentrations increased. The minnows readily degraded DEHP to 2-ethlhexyl phtahalate and phtahalic acid, with a half-life of 12.2 days.

In a continuing series of papers, Kobayashi *et al.*[255,256] reported on the accumulation, metabolism, and excretion of chlorophenols by fish. Goldfish exposed 48 hours to 15 mg/l phenol accumulated conjugated phenol in the gall bladder to a level of 2.0×10^{-6} M/g-bile compared to only 7.6×10^{-7} M/g-bile of free phenol. The isolated conjugate was identified as phenyl-β-glucuronide, indicating that biliary excretion of glucuronides is a general detoxication mechanism for fish exposed to phenolic compounds, along with the direct branchial to water excretion of phenylsulfate which was not detected in the bile. The presence of a conjugated pentachlorphenol (PCP) in goldfish bile was also ascertained after 5 hours of a 48-hour exposure to 0.1 mg PCP/l. Biliary concentration of the conjugated PCP, isolated and identified as pentachloro-β-glucuronide, increased to 4.46×10^{-6} M/g-bile in 48 hours (a concentration factor of 12 000x), whereas bile concentration of free PCP remained negligible. Pruitt *et al.*[257] investigated the toxicity, accumulation, and elimination of PCP for the bluegill. The 96-hour LC50 was 0.3 mg/l, and fish exposed to a sublethal concentration (0.1 mg/l) accumulated PCP in tissues from 10 to 350x the water concentration during the first 8 days of exposure. Thereafter, PCP tissue residues decreased, suggesting elimination or detoxification. Upon removal to PCP-free water, the fish rapidly eliminated PCP but residues ranging from 0.03–0.6 μg/g were detectable 16 days after fish were placed in a clean environment. The uptake, metabolism, and disposition of PCP and pentachloroanisole (PCA) in rainbow trout were studied by Glickman *et al.*[258] Trout exposed to 0.025 mg PCP/l for 24 hours accumulated the compound to levels of 16, 6.5, 6.0, and 1.0 μg/g in liver, blood, fat, and muscle, respectively, and concentrations of PCA in tissues of trout exposed similarly were of the same order of magnitude as was found for PCP except that fat contained as much as 80 μg/g. Differing elimination rates showed PCA to be more persistent; half-lives for PCA in the various tissues ranged from 6.3 to 23 days, while PCP had half-lives ranging from 6.2 to 23 hours. Bile from PCP-exposed trout contained high concentrations (250 μg/g) of PCP, mostly as the glucuronide conjugate. No other metabolites were detected and analyses of the tissues indicated that there was no methylation of PCP in any of the tissues studied. However, bile from PCA-exposed trout contained PCP glucuronide (10

μg/g) as well as PCA, indicating demethylation of this compound *in vivo* by rainbow trout.

Hill *et al.*[259] reported that channel catfish exposed to 2.0 mg Aroclor 1254/l for 24 hours showed maximum tissue concentrations of PCBs after 12 hours, and that these levels were maintained throughout a 480-hour, post-exposure period. An increase in hepatic microsomal activity occurred in test fish exposed for 96 hours to 1.0 mg Aroclor 1254/l; aminopyrine N-demethylase and aniline hydroxylase activities significantly increased over control fish by 68.4% and 47.5%, respectively. No inhibition of mitochondrial respiration was observed, and it was concluded that Aroclor 1254 at moderate concentrations did not interfere with the aerobic synthesis at ATP. *In vivo* inductions of aryl hydrocarbon hydroxylase (AHH) in hepatic microsomes of young coho salmon fed one of three diets containing 1.0 mg PCB/l as Aroclor 1242, 1.0 mg/l Prudhoe Bay crude oil (PBCO), or a 50-50 mixture of 1.0 μg PCB/l and 1.0 μg PBCO/l, and in fish exposed to a water-soluble fraction (WSF) of PBCO dissolved or dispersed in seawater were described by Gruger *et al.*[260] Hepatic AHH was induced in young coho exposed to 1.0 mg PCB/l, but not by 1.0 mg PBCO/l in the diet, suggesting that PCBs were predominant to petroleum hydrocarbons in inducing hepatic AHH. However, coho exposed to 0.15 mg/l WSF of PBCO for 6 days showed an induction of this enzyme system, and it was concluded that crude oil components of relatively high water solubility (for example, methylated naphthalenes and polyaklylated benzenes) may be more effective than high molecular weight insoluble components in inducing hepatic AHH.

Kreutzmann[261] investigated the side effects of chloramphenicol (CAP) and oxytetracycline (OTC), two antibiotics frequently used in fish culture, in the European eel (*Anguilla anguilla*) and concluded that OTC is preferable to CAP for therapeutic purposes on fish. Eels receiving 2 mg CAP/g body weight injected intraperitoneally showed disturbances in the production of red blood cells. These included vacuolization of the plasma, modification of the nucleus, a considerable drop in the number of erythroblasts, and disturbances in the fat metabolism of the erythrocytes. Other deleterious effects were observed in leucocytes and thrombocytes. These effects on haematopoieses were also observed after intraperitoneal injection of 2 mg OTC/g body weight, but to a lesser degree. Assessing the adhesiveness of walleye (*Stizostedion vitreum*) eggs exposed to tannin (in the form of gallotannic acid), Waltemyer[262] found that adhesiveness was proportionately reduced by 2-minute exposures to tannin concentrations ranging from 400 to 1000 mg/l. Hatching success of tannin-treated eggs was comparable to that of non-treated eggs.

In a series of papers, Hara[263] and Hara and MacDonald[264] described rainbow trout olfactory responses to amino acids, amino acid analogues, and skin mucous substances of trout and other fish species. Recordings of olfactory electrical activity established definite structure-activity relationships; those amino acids inducing olfactory stimulation were characteristically simple, short and straight-chained, with only certain attached groups (the presence of both alpha-amino and alpha-carboxyl groups was essential). Skin mucous substances induced pH dependent responses, and the response pH curves resembled those obtained with free amino acids. The active compounds, as yet unidentified but quite possibly amino acids, in mucous substances responsible for olfactory stimulation were heat stable, non-volatile, and had molecular weights between 500 and 1000.

The cytological effects of sublethal concentrations of soldium lauryl sulphate (SLS) and zinc sulphate on the interrenal and chromaffin cells of the head kidneys in goldfish were investigated by Bromage and Fuchs.[265] After treatment for up to 4 weeks with SLS at concentrations between 2 and 15 mg/l there was an increase in nucleolar size and number and more strongly defined nucleus in the interrenal cells, changes that indicated cellular activation. However, cellular activity in the chromaffin cells remained unaffected. Exposure to $ZnSO_4$ at 2 and 3 mg/l exerted a similar action to SLS on the interrenal cells, and it was suggested that the increased circulatory corticosteroid production occurring after exposure to fish to sublethal concentrations of pollutants may be important in the development of a generalized disease syndrome and to long-term success of the fish population. Abel[266] studied the effects of 10 lethal concentrations of SLS on the gills of the brown trout (*Salmo trutta*). Median survival times ranged from 45 hours at the lowest concentration (18 mg/l) to 0.07 hours at concentrations of 560 and 1000 mg/l. At concentrations to 120 mg/l SLS (Median survival times > 1 hour), pathological effects included acute inflammation of the gill tissue, extensive detachment of the epithelium and, except at the lowest concentrations, collapse of the pillar cell system. Autolysis, the formation of lysosomes and eventual dissolution of cell contents, appeared to be the

mechanism responsible for the action of SLS at concentration below 120 mg/l. At concentrations above 120 mg/l (median survival times < 1 hour), the mechanism of action was a very rapid lysis by direct action of the detergent on the cellular constituents, resulting in a complete disruption of cellular and tissue structure.

MISCELLANEOUS POLLUTANTS

Chlorine and wastewater. A study by Dickson *et al.*[267] to determine the effects of intermittent chlorination on aquatic organisms, suggested that an increased frequency of exposure resulted in increased mortality for goldfish (*Carassius auratus*). The data indicated that fewer but longer periods of chlorination might be less adverse than many short periods of chlorination. They concluded that a chlorination procedure of two or three exposures of 15 to 30 minutes per day of total residual Cl concentration, from 0.5 to 0.75 mg/l, would not result in acute lethality to fish which respond similarly to goldfish in the field. Bluegills (*Lepomis macrochirus*) were exposed by Bass *et al.*[268] to multiple pulses of free residual Cl in the water at 4 concentrations, ranging from sublethal to lethal levels (0.2 to 0.52 mg/l). Test temperatures were 15, 25, and 32°C. Rainbow trout (*Salmo gairdneri*) were also exposed to 0.41 and 0.52 mg Cl/l at 15°C until they expired. Gill hyperplasia and swelling of the lamellar epithelial cells caused blockage of respiratory gas transport across the gill epithelium and was the primary cause of death of fish exposed to several pulses of free residual Cl. Bass and Heath[269] found that Cl altered the heart rate and breathing patterns of rainbow trout. They concluded that free Cl killed fish by internal hypoxia induced by damage to the gills. In addition, behavior of juvenile bluegills exposed by Bass and Heath[270] to a series of Cl concentrations 3 times per day for 7 consecutive days at temperatures from 6 to 32C, indicated stress at 0.20 mg total Cl/l at the two highest temperatures. A threshold of 0.3 mg total Cl/l was observed, at which Cl became toxic to bluegills. The 96-hour LC50 was approximately 0.40 to 0.55 mg total Cl/l at all temperatures. At 0.52 mg total Cl/l the LT50 (median lethal time to death) decreased from 72 hours at 6°C to 20 hours at 32°C. Brooks and Seegert[271] determined that LC50 values for yellow perch (*Perca flavescens*) exposed to single 30-minute doses of residual Cl ranged from 0.70 mg/l at 30°C to 8.0 mg/l at 10°C. Triple 5-minute exposures resulted in LC50 values of 22.6 and 9.0 mg/l at 10 and 20°C, respectively. Single 30-minute exposures produced LC50 values of 0.99 and 0.94 mg/l in brook trout (*Salvelinus fontinalis*) at 10 and 15°C, respectively. Mortality of perch was delayed at higher temperatures. For both species, concentrations resulting in no mortalities were approximately one-half the LC50 value.

Spotfin shiner (*Notropis spilopterus*) and bluntnose minnow (*Pimephales notatus*) exposed to intermittent Cl discharges from a fossil fuel power plant were investigated by Cherry *et al.*[272] Clorine avoidance studies were also performed in a field laboratory. Responses of the fish to total residual chlorine (TRC) in the laboratory study varied with acclimation temperatures. Avoidance apparently occurred in the river during chlorination periods when the TRC and free chlorine residuals (FCR), respectively, were 0.46 and 0.27 mg/l or higher at river temperatures of 12°C or less. The avoidance response of spotted bass (*Micropterus punctulatus*) and rosyface shiners (*Notropis rubellus*) to TRC was found by Cherry *et al.*[273] to be closely associated with the calculated hypochlorous acid (HOCl) fraction of the TRC, which declined (relative to the percent of TRC) as pH and acclimation temperature increased. The HOCl content may be the most important constituent within the TRC to influence the avoidance response of fish in unpolluted aquatic systems. The magnitude of the Cl concentration was not as important in the avoidance study as was the relative concentration of the various Cl species that comprised the total residual.

Cohen and Denzuela[274] studied the effects of Cl on the gills of the mosquitofish (*Gambusia affinis*) after at least a 1-hour exposure and found extensive delamination of layers and separation of adjacent cells by disruption of junctions and cleavage of surface coats that couple cells together, especially epithelial and pillar. The amount of damage was concentration related and mortality corresponded to degree of gill damage. Katz and Cohen[275] used the mosquitofish to test several classes of common compounds at ambient temperature before and after excessive chlorination. Toxicity of phenol and tryptophan was increased and tannic acid toxicity was reduced by excessive chlorination. Other compounds remained unaffected. The study indicated that conditions for excessive chlorination are conducive to the formation of acutely toxic compounds at ambient temperatures. Zeitoun and Hughes[276] determined that Cl caused in-

creased concentrations of P, Mn, Fe, Cu, Zn, and K in the blood plasma of 3-year-old rainbow trout. Sodium was the only electrolyte that decreased. Chlorine toxicity appeared to disturb the mineral homeostatis in the fish blood.

Buckley[277] exposed fingerling coho salmon (*Oncorhynchus kisutch*) for 12 weeks to various concentrations of treated wastewater contained TRC with river water diluent under continuous flow conditions. Mild to pronounced symptoms of hemolytic anemia were caused by 0.03 to 0.05 mg/l of TRC. At TRC levels of 0.012 and 0.05 mg/l Heinz bodies were formed. The appearance of these bodies in fish exposed to TRC had not previously been reported. Clarke *et al.*[278] found that chlorination increased the acute lethal toxicity of Winnipeg municipal wastes to fish under laboratory conditions and to caged black bullheads (*Ictalurus melas*) in the Red River. Laboratory 96-hour LC50 values underestimated the acute lethal toxicity of the chlorinated waste, and indications were that chlorination of the municipal waste at 6 to 8 mg/l could affect the life cycle of fish at waste concentrations as low as 0.03%. Bellanca and Bailey[279] reported on two fish kills in the James River attributed to chlorinated effluents from two large wastewater treatment plants. An unusual occurrence, during both fish kills, was the large number of fish with broken backs. Subsequent laboratory studies with the effluents resulted in fish kills, but broken backs did not occur. The authors concluded that the broken back situation was associated with a chronic toxicity problem rather than an acute one. Fathead minnows (*Pimephales promelas*) and lake trout (*Salvelinus namaycush*) that had been previously exposed to sublethal levels of TRC and total residual bromine chloride in treated wastewater were found by DeGraeve and Ward[280] to have greater tolerance for the respective halogens than previously unexposed fish. A direct relationship existed between acclimation time and degree of acclimation. Kopperman *et al.*[281] reported that the disinfection of wastes resulted in the incorporation of Cl into organic molecules. Chlorinated compounds found in waste treatment effluents and their capacity to bioaccumulate in fathead minnows during chronic exposures were discussed.

Wunder[282] observed curvature in the spine and shortening of soft fins in pike-perch from Barrage Lake, which received untreated domestic wastes. Waste treatment gradually decreased the percentage of affected fish.

Pulp and paper. A broad overview of the pulp and paper mill industry effects on the biota of Nipigon Bay of Lake Superior was presented by Kelso *et al.*[283] The industry began operation in 1945. Processing was by both chemical and mechanical pulping procedures, utilizing sodium hydroxide and sodium sulphite in the lignin dissolving step. Though fish were found the least sensitive indicator of contamination effects, the once abundant stocks of fish have become limited or rare. The stocks of walleye (*Stizostedion vitreum vitreum*), the last of the desirable commercial species, have experienced a precipitous decline during 1958–67 and commercial catches were periodically rejected because of objectionable taste and odor. Movements and concentrations of fishes in association with the pulp and paper mill discharge at Red Rock in Nipigon Bay of Lake Superior were also studied by Kelso.[284] The dominant species shifted from primarily yellow perch (*Perca flavescens*), 60% of the population, with suckers second in abundance, 35%, to predominantly suckers, 92% of the population and yellow perch, only 5% in the area near the Red Rock discharge. He suggested that this was due to alterations in water quality including elevated temperatures (up to 15°C above ambient) and in benthic invertebrate abundance near but just outside the immediate discharge area. Movements of ultrasonic tagged fish released in the area of discharge showed up to 4 hours of disorientation before avoidance behavior commenced. The higher sucker concentration in the discharge area, in spite of shorter residence time per individual, was believed due to feeding response stimulated by the high benthic biomass, overriding avoidance reaction. Chlorinated bleached kraft mill effluents were analyzed by Rogers and Keith[285] using gas chromatography and GC-mass spectrometry in an attempt to identify components toxic to sockeye salmon (*Oncorhynchus nerka*) and coho salmon (*Oncorhynchus kisutch*). Only part of the acute toxicity could be assigned to known toxic constituents, trichloroguaiacol, tetrachloroguaiacol, and resin acids. It was suggested that undetected chlorinated lignins with molecular weights too small for GC analysis contributed to the observed toxicity. Servizi *et al.*[286] studied the toxicity, to juvenile sockeye salmon, of foams from two British Columbia kraft pulpmill effluents and found that the toxicity of the foams was concentrated in the methanol-soluble fractions. The 96-hour LC50 values for methanol-soluble fractions were 1.7 and 2.5 mg/l. Most of the toxicity in one plant was from neutral diterpene and

resin acid; in the other, from an unidentified nonionic surfactant pitch dispersant. The overall toxicity of the foam was 1000 times greater than the total effluents and aerated lagoon treatment had little beneficial effect on reducing foam toxicity. In examining the effects of an Ontario bleached kraft mill effluent on juvenile rainbow trout (*Salmo gairdneri*) Whittle and Flood,[287] using aerated static exposures, found tainting effects at 3 percent concentration after 48 hours while the 96-hour LC50 was 36 to 37%. Continuous-flow procedures not requiring aeration resulted in 96-hour LC50s of 21.8 to 25.8% and a 16-day LC50 at 10%. Growth reduction effects occured at 6%, about twice the concentration that resulted in tainting. They found aeration and steam stripping of the effluent reduced the toxicity of the effluent considerably and suggested that this decrease may be due to oxidation and volatilization of sulfur compounds and other volatile toxicants. The results of the Rainy River Fish Tainting Questionnaire was reported by the Minnesota Pollution Control Agency.[288] The majority of the respondents had experienced taste and odor problems with Rainy River fish and that the Sault Rapids area was most commonly associated with this problem. Sixty-eight percent of the respondents considered the problem to stem from pulp and paper mills in the International Falls area of Minnesota and Ft. Frames, Ont. Gordon and McLeay[289] reported the effects of test temperature, acclimation temperature, and species of fish on the results of the standardized sealed-jar bioassay proposed by McLeay. They found that species made a difference in the end point, using bleached kraft mill effluents (BKME). Coho salmon performed as previously predicted, using less of the available O_2 before death at higher concentrations of BKME than lower concentrations, but rainbow trout did not. In testing the effects of several variables on the results of a short-term fish-blood, sugar stress syndrome bioassay McLeay[290] reported that BKME elicited an EC50 response at 0.04 to 0.16 of the 96-hour LC50. Juvenile coho salmon and rainbow trout responded within 3 to 6 hours of exposure, with the magnitude of response greater among the coho salmon. Liver glycogen reserves in the test fish were directly related to the level of blood sugar elevation following exposure. When fish were exposed to effluent after foam separation treatment the toxicity response was eliminated. When exposed to effluent after only biotreatment the response levels were considerably reduced but still above control values.

Using growth and production of chinook salmon (*Oncorhynchus tshawytscha*) in laboratory streams Seim *et al.*[291] studied potential effects of kraft mill effluents and stabilized kraft mill effluents on aquatic ecosystems. Season of the year, the source of the effluent, concentration of the effluent, fish stocking rate, and degree of treatment affected the results. Reduced fish production was experienced in streams receiving as low as 1.5% kraft mill effluent. Concentration as high as 4% stabilized kraft mill effluent produced increased production. Increased production in streams receiving up to 4% stabilized kraft mill effluent was believed due to enrichment-stimulated food production in the absence of toxicity.

Radioactive. Egami and Hama[292] exposed adult medaka (*Oryzias latipes*) to ^{60}Co irradiation at nine exposure rates from 0 to 360 R/day. Median survival times were inversely related to dose rates; 60 days at 360 R/day, 90 days at 160 R/day, 150 days at 90 R/day, and no detectable effects at 10 R/day or less. When irradiation survivors were mated to untreated mates hatchability of embryos was reduced; however, dose-rate effects were not clear in this portion of the tests. Hyodo-Taguchi[293] also exposed adult male medaka to ^{60}Co irradiation at dose rates of 3.3 to 120 R/day for 30 days during the breeding season. Fish irradiated at 17 and 80R/day, when observed after 30 and 17 days of exposure, respectively, had gonad-somatic indexes reduced to about half of the controls. After 10 days of exposure, spermatogonia Ib numbers were down from 25 to 91% of control values when exposures ranged from 3.3 to 127 R/day, respectively. After 30 days of exposure, spermatogonia Ib numbers were higher than at 10 days among groups exposed to 3.3 to 6.7 R/day, down 6 and 42% of control values, respectively. At 17 R/day and greater, 30-day spermatogonia Ib numbers were down to 22 to 0% of controls. Spermatogenetic activity of medaka exposed to ^{60}Co at 3.6 through 360 R/day was studied by Michibata and Egami.[294] Spermatogonia Ad was observed to be very sensitive to gamma radiation with none of these cells present following exposure at about 3 kR, at the rate of 9, 160, and 360 R/day. Exposures of 6 kR at 23 and 40 R/day resulted in reductions of spermatogonia Ad cell counts of about 20 and 40% of control values, respectively, with ovotestes frequently observed.

Pechkurenkov[275] reported that increased chromosomal aberrations in fish embryos might be expected at a dose rate of not less

than 1 rad/hour. Conclusions were based on fish embryos grown in ^{90}Sr-^{90}Y solutions. Edmundson[296] reported that there were no significant effects of temperature, 10°, 15°, and 20°C, and/or gamma radiation 0, 320, 560, 870, and 1000 rads on growth of juvenile rainbow trout (*Salmo gairdneri*) for 4 weeks following irradiation. Chee[297] exposed fathead minnows (*Pimephales promelas*) to ionizing radiation at the rate of 37.8 rad/minute and found that LD50 to be 2650 rads in 30 days and determined that dose rates had a strong influence on the resulting LD50. Fathead minnows were then exposed to sublethal doses of radiation up to 30% of the LD50 dose to determine predator-prey relationships between irradiated and non-irradiated fish using largemouth bass (*Micropterus salmoides*) as a predator. No significant correlation was found between fathead minnow survival rates and irradiation doses within the limits of the experiments.

Hewett and Jefferies[298] studied the accumulation of ^{137}Cs by brown trout (*Salmo trutta*) and reported ^{137}Cs concentration factors from the water for nine organs and whole fish after exposures at intervals from 1 day through 420 days. Whole fish concentration factors ranged from 0.132 on the second day of exposure to 4.96 on the 420th day with a steady state reached between the 100 and 200th day. Higher concentration factors were found in larger fish which was believed due to diet. Spigarelli[299] tagged chinook salmon (*Oncorhynchus tshawytscha*), rainbow trout, and brown trout with thermal dose accumulating tags to study the effects of thermal plume residence on the ^{137}Cs uptake of Lake Michigan fishes. He concluded that thermal plume resident brown trout had higher ^{137}Cs residues than non-plume resident fish but that this was not demonstrable in chinook salmon and rainbow trout. It was suggested that the ^{137}Cs came from the water-plankton-forage fish-salmonid food chain and that higher levels of plume resident fish was due to the increased metabolic rate induced by the higher temperatures in the heated plume. Fish inhabiting a cooling lake for a power reactor were sampled by Shearin and Lyon[300] to determine tissue residues of radioisotopes. Concentrations of radioactivity varied considerably from species to species and from one sampling time to another. Samples contained ^{137}Cs, ^{58}Co, ^{60}Co, ^{51}Cr, and ^{54}Mn, usually in the 0 to 500 pCi/kg range.

Culinary practices were shown by Marej *et al.*[301] to have a major influence on the amount of radioisotopes (^{137}Cs, ^{90}Sr, ^{65}Zn, and ^{60}Co) in human diets composed of contaminated fish. It was reported that radioisotopes were at a minimum when the fish were boiled, particularly when the water used was changed during cooking; the boiling extracted the contaminated minerals during processing. Frying had the least beneficial effect in removing the contaminants and fish broth was found high in contaminants. Brining also caused contaminants to pass from the fish into the brine reducing dietary radioisotope concentrations. Bakunov Garanina[302] found ^{90}Sr and ^{137}Cs levels in Caspian Sea fishes to differ between fish species, and ^{90}Sr was found to be more concentrated in tissues of freshwater species than among marine species and ^{137}Cs more concentrated in tissues of marine fishes. Fulk'e and Grobi[303] studied the dynamics of the accumulation of ^{65}Zn by eels from freshwater and found that 30% accumulated in muscles, 15% in skin and mucus, 10% in digestive canal, 9% in skeleton, and 8% in the gills. Exposure to ^{65}Zn at 7°C resulted in lower accumulation than when exposed at 18 and 23°C. Alberts *et al.*[304] studied the uptake of ^{238}Pu in seven species of freshwater fishes from an area contaminated by a spill in 1969. In all species the tissue residue concentrations were highest in the GI tract, gills, and liver; intermediate in concentration in the bone and skin; and least concentrated in the muscle and gonad tissues. Because of the consistently low ^{238}Pu concentration in the muscle, the authors speculated that there was little likelihood of Pu transfer to man through this pathway.

Other. Mukherjee and Bhattacharya[305] exposed the air breathing fish *Ophiocephalus punctatus* and *Clarias batrachus* to sodium sulphide, phenol, Cu, ammonia, and a combined effluent. The data indicated that the low dose of pollutants that produced only 10% death was no less physiologically effective than the LC50 doses. Verma and Dalela[306] determined the toxicity of distillery wastes to *Puntius sophore* and *Mystus vittalus*. Survival rates were dependent upon concentration, time of exposure, and temperature. The acute toxicity of 47 industrial chemicals to the bluegill (*Lepomis macrochirus*) and the tidewater silversides (*Menidia beryllina*) was studied by Dawson *et al.*[307] Twenty-nine of these substances were clearly hazardous under proposed criteria. Three of the 18 remaining had freshwater LC50 values in the 550 to 650 mg/l range but were toxic below 500 mg/l in salt water and were also hazardous. The remaining 15 substances were clearly above the proposed criteria. Maylin *et al.*[308] determined

the toxicity of laundered fabrics treated with the following flame retardants: Antiblaze 19 (a mixture of cyclic phosphates), Fyrol 76 (an oligomeric vinyl phosphonate), Pyrovatex CP (N-methylol dimethyl phosphonopropioamide), TDPBPP (trix (2,3, dibromoprophyl) phosphate), and THPOH (tetrakis (hydroxymethyl) phosphonium hydroxide). Immersion of laundered or unlaundered flame retardant 100 percent polyester or polyester blend fabrics used in children's sleepwear in water containing goldfish (*Carassius auratus*) resulted in the release of TDBPP (tris (2,3-dibromopropyl) phosphate) and death of goldfish within 24 hours. TDBPP undergoes a loss of HBr in water and production of a metabolite. The fish responded identically to those held in water to which 1 mg pure TDBPP/l was added. The other flame retardants did not indicate acute toxicity to the goldfish. Nusch[309] evaluated the acute toxicity of Cu chelated with citric acid, NTA, and EDTA with bacteria, algae, protozoans, lower metazoans, and fish. Strong chelators such as NTA and EDTA had a detoxifying effect of one to three orders of magnitude compared with the toxicity of unchelated Cu. Toxicity tests with Cu-citrate-complexes indicated 1 mg Cu/l was more toxic than 100 mg/l when chelated with equimolar concentrations of citric acid.

PHYSICAL FACTORS

Wiley and Dufek[310] found 6.2 times as great a trout biomass inhabiting a 650-ft section of non-modified Wyoming mountain trout stream as in a similar section of channelized stream. Cover was the primary habitat loss due to channelization. Stream channelization and straightening were also found by Huet and Timmermans[311] to adversely affect fish population in several ways. Shortening the stream length between two points increased the gradient and thus species not adapted to increased water velocity were eliminated. Loss of original length also resulted in a reduced productive area. Fish losses associated with these changes amounted to 20 to 60% of the species, depending on the severity of the straightening. Losses were nearly complete when all vegetation was eliminated by riprapping the banks, lesser losses were experienced when varying amounts of submergent vegetation was eliminated. Siltation also associated with channelization added to the decline in fish population. Bulkley *et al.*[312] summarized the finding of five separate subprojects entitled "A Study of the Effects of Stream Channelization and Bank Stablization on Warm Water Sport Fish in Iowa." The reports on these subprojects were: Subproject I. Inventory of Major Stream Alteration in Iowa, by R. V. Bulkley[313]; Subproject II. A Study of the Impact of Selected Bank Stabilization Structures on Game Fish and Associated Organisms, by A. L. Witten and R. V. Bulkley[314]; Subproject III. Some Effects of Short-Reach Channelization on Fish and Fish Food Organisms in Central Iowa Warm Water Streams, by L. R. King and K. D. Carlander[315]; Subproject IV. The Effects of Long-Reach Channelization on Habitat and Invertebrate Drift in Some Iowa Streams, by D. Zimmer and W. Bachmann[316]; and Subproject V. Effects of Long-Reach Stream Channelization on Distribution and Abundance of Fishes, by B. W. Menzel and H. L. Fierstine.[317] The overall findings of these studies were that between 1600 and 4800 km of stream, capable of supporting permanent fish populations, were lost in Iowa through stream channelization and what remained of the channelized sections had reduced numbers of fish species due largely to reduction in habitat diversity. Wherein, brush piles and trees had accumulated during the 10 to 15 years since channelization had occurred fish were in some areas as abundant as in natural sections. Instream and bank-stabilization structures that contributed to increased habitat diversity contributed to increased numbers of fish and fishes. Golden and Twilley[318] reported reductions in stream length, biomass of fishes, and numbers of species in Big Muddy Creek, Ky. persisting 33 years after channelization. Effects were most noticeable among game fish species. Adverse effects were not considered due to water quality but rather due to removal of submerged cover and uniformity of stream flow within the channelized area. More than 52 years have failed to return a channelized segment of the Luxapalila River (Mississippi-Alabama) to productive levels equal to an unchannelized segment according to Arner *et al.*[319] Largemouth bass (*Micropterus salmoides*) in the unchannelized segment averaged eight times larger than those in the channelized segment but were normally distributed in size whereas those in the channelized segments were mostly in the smaller size classes. Largemouth bass were also more common in the unchannelized segment. Adverse effects of channelization were considered to be primarily due to general uniformity of habitat in the channelized zones and lack of firm substrate, water quality differences between channelized and unchannelized segments were reported to be insignificant. Immediately downstream from a highway culvert

construction site on a small Ontario trout stream total numbers of fish were observed by Barton[320] to decline from 27 kg/ha before construction to 10 kg/ha following construction. These changes were not observed further downstream nor did they persist following completion of construction activities. Replacement of riffle area by addition of rubble and boulders and removal of construction sediments were discussed as means to improve recovery of biota. A discussion of effects of accumulated large organic debris (logs, limbs, tree tops, and root wads) on the instream environments and potential changes that could follow removal of this debris were presented by Swanson *et al.*[321] Such debris was considered to have both adverse and beneficial effects on salmonid stocks of western Oregon streams. Adverse effects included blocked fish passage, siltation of spawning sites, streambank erosion, and instability of spawning gravel with potential losses of embedded eggs and fry. Beneficial effects included stabilization of streambeds and banks, slowing of movement of fine organic matter downstream, promotion of more complete *in situ* biological processing, and enhancement of habitat diversity. The extent of fish losses, particularly losses of five species of Pacific salmon, due to dredging activities in the Lower Fraser River of British Columbia, Canada was studied by Dutta.[322] At least 98.8% of the dredging-entrained fish died within 96 hours of passage and recommended control of dredging during peak downstream migratory periods.

After exposing striped bass (*Morone saxatilis*) and white bass (*Morone americana*) embryos and larvae to different shear forces in rotating laboratory apparatus Morgan *et al.*[323] estimated 1-, 2-, and 4-minute median lethal shear (LS50) values. It was pointed out that damage may result from deformation, rotation, or both. Damage was primarily seen as yolk aberrations such as breakup of yolk droplets or separation of the yolk from the embryo. Embryos of both species had 1-minute LS50 values from 425 to 542 dynes/cm^2 and 4-minute LS50s of 175 to 190 cynes/cm^2. The LS50s reported are well below forces induced by passage of ships through spawning areas but are close to but below those experienced in passage through cooling water systems in some power generation stations, provided no physical contact is made with moving parts of pumps or the walls of conduits. The effect of acute hydrostatic pressure changes of 0 to 50 atmospheres on larvae and juvenile fishes were studied by Lampert.[324] Pressure changes of this magnitude were implicit in a Rhine River pumped storage facility where fish had been found in the reservoir without known introductions. Fish responses were suggestive of gas bladder volume loss, with fish quiescent on the bottom of the recovery vessel for a time before recovery to apparent normal swimming. All recovered within 2 hours, more than half within 5 minutes. Perch (*Perca fluveatilis*) were the last to recover, some requiring in excess of 60 minutes. Whitefish and small carp (*Cyprinus carpio*) showed no apparent ill effects. Simmons[325] summarized findings from a number of studies on the Smith Mountain-Leesville Project and stated that comprehensive studies on effects of the pumped storage project are still needed, but that knowledge to date does not indicate any adverse effects on the fisheries. A short addendum to the paper stated that the endemic centrarchid species have adjusted their spawning depth to compensate for pumped-storage-induced water level fluctuations of the order of 1.8 to 2.3 m/week. Robbins and Mather[326] reported no detectable adverse effects of the Muddy Run Pumped Storage Project on the fish populations of its water source, Conowingo Pond, on the lower Susquehanna River. However, water level fluctuations in Muddy Run in excess of 15 m/week with weekly refilling severely inhibited reproduction of nest building fish species. As a consequence, recruitment of these species was by entrainment from Conowingo Pond. Water level fluctuations in Conowingo Pond were usually less than 1 m/day due to greater total volume and replacements from upstream. Mechanical damage of some specimens was reported but the significance of these losses were believed insignificant in spite of the fact that 6.5 times as many fish larvae were pumped into Muddy Run as left that pond during the generation phase of the cycle. Generalities related to potential impact of pumped storage operations on fishes were discussed by Hauck and Edson.[327] This type of project is becoming more common in the total electric generation output of the nation. The impact on fishes was physical damage due to entrainment in the pumped water, sometimes with fish making multiple passes through the system and flooding and dewatering of aquatic habitats as the pumping and discharge reversals of flow occurred. Needs for more research on reducing damage to fish passing through turbines and pumps was stressed, as well as for research in reducing entrainment itself.

Walburg[328] reported fish population changes over the period 1956 to 1974 as the area now occupied by Lewis and Clark Lake changed

from main stem Missouri River to reservoir habitat. Over this period the net result has been a decline in adult fish catches of approximately 66%, with the non-game species affected most. Species reductions have also been observed, from 66 species in the 1956–57 catches to only 28 in the 1972 to 1974 period. Spawning success and recruitment reductions from siltation, water level fluctuation, reduced spring flooding, and washout of young fish at high relative discharge rates were responsible for most losses. Loss of forage fish resulted in reduced growth rates of predator species. Changes in the fish community of the delta zone following hydro-electric damming of the Volga River were studied by Orlova and Popova.[329] Work was centered on the responses of pike (*Esox lucius*) and the sheatfish (*Silarus glanis*). The pike declined in numbers while the other has prospered. The most important reasons for these shifts were considered the reduced and delayed high water period. These conditions adversely affected recruitment of the spring-spawning pike, spawning success, and the foraging period of the young. Other spring-spawning fish were similarly affected. However, the sheatfish, a summer spawner, was favored by more stable summer water levels. The effects of impoundment of Big Walnut Creek, Ohio by Hoover Dam was described by Cavender and Crunkilton.[330] Changes in fish populations were considered due primarily to inundation of the stream channel, prevention of migration, and alteration of water velocity, substrate, turbidity, temperature, and O_2. Standard and new techniques of statistically treating the data to evaluate differences were presented. Changes in fish populations in the Middle Zambezi River following the first 8 months of impoundment were studied by Jackson and Rigers.[331] He described rapid population shifts from lotic-shallow water species to comparatively high numbers of small migratory fishes. These changes were not considered long lasting. Kushlan[332] reported changes in an Everglades fish community when an unusual wet season provided continuous, abundant water in a system accustomed to an annual loss of surface water. The resultant 27-month stable, high water levels superimposed indigenous large carnivorous fish on a community previously dominated by small omnivorous species better adapted to the periodic loss of surface water. This shift in dominant controlling factors from seasonal surface water scarcity to that of predation by a few large carnivores resulted in fish density decreases, but promoted increases in biomass, mean fish size, species richness, and species diversity. A detailed hydrologic-geologic study of the Ash Meadows area in southern Nevada, as related to irrigation pumping and loss of the rare desert pupfish (*Cyprinodon* sp.) habitats, was reported by Dudley and Larson.[333]

Both reduced light intensity and increased turbidity were reported by Vinyard and O'Brien[334] to substantially diminish the reactive distance of bluegill (*Lepomis macrochirus*) for all prey sizes. It was suggested that these factors may have significant implication on feeding rates of this species in natural habitats. The influence of turbidity and light on the feeding activity of two species of fish, the bluegill, a visual feeder, and the gizzard shad (*Dorosoma cepedianum*), a non-visual feeder, was studied by O'Brien.[335] Both light reduction (as a result of turbidity) and turbidity itself reduced the capacity of the visual feeder to find and thus obtain food. Because these factors were not expected to inhibit feeding by the shad they were considered to have a competitive advantage over the bluegills under turbid conditions. This suggested a reason why shad abundance is correlated with turbidity in natural waters. Cherry *et al.*[336] studied an environment receiving power plant effluents including heat as high as 44.5°C and coal ash turbidity to 275 JTU and found temperature more limiting than turbidity and coal ash effects greatest in terms of contributions of heavy metals to tissue residues. The conditions limited fish populations to mosquitofish (*Gambusia affinis*) only. Of the 40 elements studied by neutron activation analysis only Ca, cloride, Se, Zn, and bromide were concentrated by the fish. Sediments from the Duluth-Superior Harbor were used by Prater and Anderson[337] as substrates in a 96-hour recycling-flowing-water test system in which the mayfly (*Hexagenia limbata*), the isopod (*Asellus communis*), *Daphnia magna*, and the fathead minnow (*Pimephales promelas*) were exposed. In all cases the fathead minnow was as tolerant or more tolerant than the other species. Common carp and rock fish (*Sebasticus marmoratus*) were examined by Sakaguchi *et al.*[338] for damage to internal and external organs following underwater explosions. Rupture of the sinus venosus and damage to the liver and kidney were the most common effects.

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